STUDY ON ETIOPATHOGENESIS, CLINICAL PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS

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In partial fulfillment of the regulation for
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CERTIFICATE

This is to certify that this dissertation titled **"STUDY ON ETIOPATHOGENESIS CLINICAL PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS"** is a bonafide work done by **Dr.S.SENTHUR RAJA PANDIAN**, Department of NEUROLOGY, Government Rajaji Hospital and Madurai Medical College, Madurai under my direct guidance and supervision in partial fulfillment of the regulations of The Tamilnadu Dr.M.G.R.Medical University for the award of D.M**, Branch I (Neurology)** during the academic period of **August 2011 to August 2014.**

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DECLARATION

I, **Dr. S.SENTHUR RAJA PANDIAN** solemnly declare that the dissertation titled **"STUDY ON ETIOPATHOGENESIS CLINICAL PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS "** has been prepared by me. I also declare that this bonafide work or part of the work was not submitted by me or any one for any degree or diploma to any other university board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of **D.M., Branch I (Neurology)** to be held in **August 2014.**

Place : Madurai **Dr.S.SENTHUR RAJA PANDIAN**

Date :

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CONTENTS

STUDY ON ETIOPATHOGENESIS, CLINICAL PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS

ABSTRACT:

SETTING: Department of Neurology**,** Government Rajaji Hospital, Madurai, Tamilnadu.

OBJECTIVES: To study 100 cases of completed stroke patients in the age group < 45 years with regard to 1. Etiopathogenic risk factors analysis, 2. Clinical presentation of young stroke 3. Their outcome in further follow up.

DESIGN: Single Observational study**.** This study was conducted among 100 Young stroke patients who were admitted in our hospital with various etiologies since March 2013 to February 2014.

RESULTS & CONCLUSION Stroke under 45 yrs is common in the age group between 35-45 years. Males are affected more than females in young stroke. Ischemic stroke more common than hemorrhagic stroke. Thrombotic stroke more common than embolic stroke in ischemic strokes

Anterior circulation strokes more common than posterior circulation strokes. MCA more commonly involved than ACA and PCA. Motor weakness is the common presentation of young stroke followed by unsteadiness, unconsciousness and speech disturbances. Right sided weakness more common than left sided weakness. Hemiparesis more common than hemiplegia. Motor aphasia most common type of aphasia in our study. Smoking and Alcohol are the most common risk factor for ischemic stroke followed by Dyslipidemia, Diabetes mellitus and Hypertension. Atrial fibrillation is the most common risk factor for embolic stroke. Rheumatic heart disease is the most common cardiac disease causing embolic stroke. Hypertension is

the single most important factor for hemorrhagic stroke. Vasculitis,Hypercoagulable states, Homocystinemia, APLA are rare risk factors for young stroke. Weakness and speech disturbances were improved significantly. Unsteadiness not improved significantly. Death were seen in 12 cases

Keywords: TIA, MCA, ICH, MRS

INTRODUCTION

Stroke is the leading cause of death and disability world wide. Stroke is a morbid life threatening condition that requires rapid and aggressive treatment to the progression from cerebral ischemia to cerebral infarction.

In developing countries like India, the incidence of stroke under 45years is increasing. Stroke in young age is relatively uncommon when compared to old age but has serious impact on the affected family as well as society. The causes are more diverse and differ as compared to the elderly. Overall prognosis is better than elderly but there is still significant morbidity and mortality.

Knowledge of the risk factors that leads to stroke under 45 years and evaluation of them is necessary for better primary prevention and health care.

AIMS AND OBJECTIVES OF THE STUDY

To study 100 cases of completed stroke patients in the age group < 45 years with regard to

1. Etiopathogenic risk factors analysis
2. Clinical presentation of young stroke
3. Their outcome in further follow up.

REVIEW OF LITERATURE

Stroke : Definition :

WHO defines stroke as rapidly developing clinical signs of focal disturbances of cerebral function lasting >24 hrs or leading to death with no apparent cause other than vascular origin.

Risk factors for ischemic stroke :

A number of risk factors that may be classified as modifiable and non modifiable increases the risk for stroke.

Non modifiable	Modifiable
Age	Hypertension
Gender	Transient Ischemic Attack
Race / ethnicity	Prior stroke
Family history	Asymptomatic carotid artery stenosis
Genetics	Cardiac disease
	Diabetes mellitus
	Dyslipidemia
	Alcohol consumption
	Increased fibrinogen
	Elevated homocysteine
	Low serum folate
	Elevated cardiolipin antibody
	Obesity
	Oral contraceptive use

Non modifiable risk factors:

Age :

The incidence of stroke gradually increases with age increasing. Data from the Framinghom study shows that incidence of stroke rises from 2-5/1000 in 45-55yrs, 10/1000 in 56-65 yrs, 20/1000 in 65-85 yrs.

The incidence of stroke under 45 years is 2 in 10000.

Sex :

In the Framinghom study(1) men aged 45-55 years have a stroke incidence rate of 20/10000, women aged 45-55 years have a stroke incidence rate 11/10000. Man develops strokes at a higher rates than woman up to the age of 75 yrs.

Race and Ethnics :

The rate of incidence of stroke in blacks higher than white. Blacks had been thought to have higher rates of intracranial atherosclerotic occlusive disease. Auckland, Newzealand, Pacific island people have higher mortality with in 28 days of stroke when compared to Europeans.

Chinese, Koreans, Japanese have higher incidence of intracerebral hemorrhage when compared to whites.

In developing countries(2) like India, the average age of patients with stroke is 15 years younger than that in developed countries(3). In India, nearly one fifth of patient with first ever stroke admitted to hospitals are under 45 years.(4) Trivandrum stroke registry showing stroke of about 9.5% under 45 years(5).when stroke occurs in the main income earner in the household there may be enormous consequences for the welfare of the family(6).Indian studies have shown that about 10to15% strokes occur in people below the age of 40 years(7).

Heredity :

Heredity seems to play a minor role in the pathogenesis of cerebral infarction. However an increased risk is seen with family history of stroke among first degree relatives. Number of inherited disease like Ehler danlos syndrome, Marfan syndrome, Osler Rendu Weber disease, Sturge weber syndrome, Hereditary dyslipoprotenemia, Deficiency of Protein C, Protein S, Antithrombin III, MELAS, Fabry's disease, Homocystinuria can cause stroke.

Modifiable Risk factors :

Hypertension:

According to Chobanian et al 2003.

	Systolic	Diastolic
HT	> 140 mmHg	> 90mm Hg
Pre HT	120-139mmHg	80-89mmHg
Optimal BP	< 120	< 80mmHg
Stage I	140-159	90 – 99
Stage II	> 160	> 100

In India many patients with hypertension are under treated or untreated.

Hypertension predisposes to ischemic stroke by aggravating atherosclerosis and accelerating heart disease. Increasing the relative risk for stroke an estimated 3-4 fold.

The risk is greater for patients with isolated systolic hypertension and elevated pulse pressure. Lowering blood pressure is stroke survivors helps prevent recurrent stroke. BP reduction in systolic BP of 10 to 12mmHg and 5mmHg diastolic BP is associated with 33% reduction in stroke incidence.

Data from Framinghom(1) study shows that men and women with definite hypertension have a three fold greater risk of stroke than normotensive individuals.

Female with hypertension who use oral contraceptive pills has 10 fold increased risk of stroke compared with female nonusers of oral contraception pills without hypertension.

Diabetes Mellitus :

Diabetes increases the risk of stroke an estimated 3 to 4 fold as compared with risk in people without diabetes. In addition diabetes increases mortality and morbidity after stroke. The mechanism of stroke secondary to diabetes caused by accelerating atherosclerosis, cardiac embolism, rheological abnormalities, low HDL and increasing platelet adhesiveness. Diabetes associated with hypertension adds significantly to stroke risk(9). Diabetic patients with autonomic neuropathy, retinopathy appear to be a group particularly high risk for ischemic stroke. High insulin level increases the risk for atherosclerosis and may represent a pathogenic factor in cerebral small vessel disease(10). Recent data suggests that aggressive management of

glucose levels in ICU care unit is associated with decreased mortality and morbidity. (11)

In most diabetes atherosclerosis begin to appear whatever their age, with in few hours of the onset of diabetes mellitus. 70% diabetes under 40 years have moderate to severe atherosclerosis.

In diabetes atherosclerosis tend to be numerous and florid, tend to undergo ulceration, clacification, and superimposed thrombosis.

Dyslipidemia :

High total cholesterol and high low density lipoprotein are correlated with atherosclerosis. Recent meta analysis however have suggested that ischemic stroke increases with increasing cholesterol and reduction in stroke risk associated with statin therapies is related to reduction in LDL.(12) Current guideline The American heart Association and proposed modifications of the NCEP III guidelines would therefore suggests that all patients at risk for stroke or who had cerebral infarction should be treated to goal LDL level less than < 70mg/dl (13).

Smoking :

Smoking is a biologically plausible independent determinant of stroke. Smoking has been associated with 70% increased risk of stroke. Stroke risk was greatest in heavy smokers and reduced within 5 years among those quit. It is also a independent determinant of carotid artery plaque thickness, intra cerebral hemorrhage, overall the stroke risk attributed to cigarette smoking is greatest for subarachnoid hemorrhage, intermediate for cerebral infarction and lowest for cerebral hemorrhage. There is even some excess risk in passive smokers.

Alcohol :

Alcohol consumption has been shown to be a risk factor for both intracranial and subarachnoid hemorrhage. The relative risk of stroke increased with heavy alcohol consumption (five or more drinks / day) and decreased with light drinking when composed to non drinkers. Heavy alcohol consumption induces cardiac arrhythmias, cardiomyopathy and hypertension increases clotting factors, increases platelet aggregation, also causes activation of sympathetic nervous system. All these factors can lead to stroke. In addition acute increase

in blood pressure can occur during alcoholic binges and frequently associated with hemorrhagic stroke.

Cardiac Disease :

Presence of Cardiac disease is the most common etiology for embolic stroke. In the Framinghom study only 13.6% of patients were free of any heart disease, 80% were hypertensive, 32.7% had prior coronary artery disease 14.5% had cardiac failure, 13.5% had Atrial fibrillation. And this study also showed the relative risk as 1.9 for men and 2.2 for women.

Coronary artery disease is an indicator of diffuse atherosclerotic vascular disease and act as a potential source of emboli from mural thrombi due to congestive heart failure, dilated cardiomyopathy, myocardial infarction. Hypertensive heart disease is associated with an increased risk of thrombo embolic and hemorrhagic stroke and peripartum cardiomyopathy and alcoholic cardiomyopathy are associated with thrombo embolic events. Literature is available suggesting that risk of coronary artery disease is higher in young Indians(14).We know that risk factors for stroke and CAD are same(15).

Atrial fibrillation :

It is the single most important factor for thrombo embolic stroke. Atrial fibrillation due to rheumatic valvular heart disease is the strongest association increasing stroke by seventeen times. Non valvular atrial fibrillation and lone atrial fribrillation also increases stroke risk especially with advancing age. The prevalence of atrial fibrillation increases with advancing age 5% in 50-60 yrs, 8.8% in 80-90 yrs. Heart failure, hypertension, diabetes, prior stroke, TIA, Age > 75 yrs increases the risk of embolism in patients with non valvular atrial fibrillation.

Obesity :

The prevalence of obesity has increased throughout India. Body mass index > 30 or and abdominal obesity are important risk factors for coronary heart disease and stroke. So regular exercise lowers arterial blood pressure, decreases insulin resistance and increases HDL, associated with low cardiac and stroke risk.

Atherosclerotic lesions :

Atherosclerotic lesions of the carotid artery bifurcation are a common cause of stroke. Asymptomatic carotid artery disease carries a greater risk for stroke. It carries the risk of stroke 1.5% at 1 yr and 7.5 at 5 yrs. Asymptomatic carotid artery stenosis of < 75% caries a stroke risk of 1.3% annually, stenosis of > 75% carries a risk of 10.5% per year. Plaque structure rather than degree of carotid artery stenosis may be a critical risk factor for stroke. Ulcerated, echolucent, and heterogenous plaque with a soft core represent unstable plaque at high risk for producing arterio arterial embolism.

Transient Ischemic Attack :

Patients who suffer TIA are greater risk than normal controls for stroke. The risk for stroke is approximately 3 times higher. Approximately 10 to 15% of those experiencing a stroke have TIA before their stroke. The interval from the last TIA is an important predictor of stroke risk. Of all patients who subsequently experience stroke, 21% do so in 1 month, 51% do so in 1 year of the last TIA. According to Johnson et al 2000 50% of those strokes following a TIA occurred within 48 hours of TIA onset (16). Patients with TIA and DW

MRI lesions are at greater risk of experiencing a subsequent stroke man those patients without a lesion(17).Older patients with isolated vertebro basilar symptoms and a significant history of cardio vascular risk factor should however be evaluated the possible TIA or stroke.(18).

Homocystinemia :

Elevated plasma homocysteine level is an independent risk factor for atherosclerotic disease. Patient with high homocysteine level increased risk of thrombotic stroke and PVD in family in young patients. Metabolism of homycysteine requires pyridoxine, cobalamine, folate, and betaine. Plasma homocysteine concentrations may be reduced by the administration of folic acid alone or in combinations with Vit B6 and Vit B 12. Conversely serum folate concentrations less than or equal to 9.2Nm have been associated with elevated plasma level of homocysteine. And decreased folate concentration alone may be a risk factor for ischaemic stroke particularly among blocks(19).

Anti Phospholipid Antibody Syndrome:

Antiphospholipid antibodies are marker for an increased risk of thrombosis including TIA and stroke particularly in younger patients. The presence of antiphospholipid either lupus anticoagulant or

anticardiolipin antibody is confirmatory test for APLA. Antiphospholipid syndrome associated with presence of APL antibodies in high titre have pathogenic role in arterial and venous thrombosis. Ischemic stroke was the most common presentation in 1000 patients (20). There is consistant association between young ischemic stroke and presence of lupus anticoagulant and anticardiolipin antibodies(21).With regard to the increased incidence of young stroke among patients with SLE found to have antiphospholipid antibodies. Lupus alone is associated with an increased incidence of cerebro vascular events. The component required for ACL binding is B2 glycoprotein 1 (B2 GPI). It is the B2GP1 dependent ACL of the IGG isotype that has been significantly associated with stroke (22).It also associated with ocular ischemia, cerebral venous thrombosis, migraine, dementia, chorea, and transverse myelopathy.

Patent Foramen Ovale :

The potential link between PFO and young stroke remains controversial. PFO is however a more common finding among young patients who present with cryptogenic stroke (23).The concurrent presence of an Atrial septal aneurysm found in 2.2% of the population

likely adds further risk(24).Paradoxical embolism caused by Right to Left shunt through PFO or ASA can be responsible for stroke. Antiplatelets, oral anticoagulants, trans catheter or surgical closure of PFO have been recommended.(25).Coexistence of PFO and ASA increases the risk of embolic stroke(26).

Migraine :

The evidence from case control studies suggest that migraine particularly migraine with aura is associated with increased risk of ischemic stroke in young women under 45 years of age (27). The patho physiological mechanism underlying this remains unclear. Rarely stroke occurs secondary to cerebral hypoperfusion during the aura phase is remain the main theory. True migranous infarcts are probably rare and tend to affect the posterior circulation. Migraine as a risk factor for future ischemic stroke seems to apply mostly in women(28) and the relative risk may be high fold in those experience migraine with aura. Childhood migraine can also cause stroke in children(29)

Migraine patients had increased risk of subsequent total stroke and ischemic stroke compared with those not reporting migraine(30).

Cerebral infarcts complicating migraine are mostly cortical and involve the distribution of posterior circulation.

CADASIL:

Cerebral autosomal dominant arteriopathy with subcortical infarct and leuco encephalopathy is a familial non arteriosclerotic, non amyloid angiopathy characterized by migraine with aura, recurrent ischemic strokes leading to pseudo bulbar palsy, cognitive decline, subcortical dementia, and white matter Hyperintensities on MRI. CADASIL is caused by simple missense mutation in Notch 3 gene on chromosome 19q12. Pathologically there is characteristic granular osmophilic material in arterial walls including dermal arteries(32).

Familial hemiplegic migraine characterized by transient weakness or frank paralysis during the aura, has also been mapped closed to the CADASIL locus(33).

Non atherosclerotic vasculopathies :

Carotid artery dissection, Migraine, Vasculitis, Radiation vasculopathy, cerebral autosomal dominant arteriopathy with subcortical infarct and leucoencephalopahty (CADASIL), Mitochondrial Myopathy, Encephalopathy, Lactic acidosis and Stroke

like episodes (MELAS), Reversible vasoconstriction syndrome, Moya Moya disease, Sneddon syndrome, Fabry's disease and malignancy all come under the hedding of non atherosclerotic arteriopathy. The most common of those in young stroke patient is cervical artery dissection which has been implicated in upto 20-25% of cases of young stroke followed by vasculitis, Infection and Moyamoya in Asian population.

Carotid artery dissection :

It accounts for upto one fifth of ischemic stroke in young and middle aged patients (34). In majority of cases the specific etiology remains unknown. Trauma, Infection Migraine, Ehler danlos syndrome, Fibro muscular dysplasia, familial(35) are the main causes of dissection.

Oral contraceptive pills :

With regard to stroke in young women OCP use is associated with a 2to5 fold increased risk of stroke.(36) It causes ischemic stroke by causing increasing blood viscosity, alteration of vessel wall with intimal hyperplasia. There are decreased levels of protein S, antithrombin activities and plasminogen activator content in women taking oral contraceptive. Oral contraceptive may also enhance arterial

hypertension. One study showed that oral contraceptive containing 30 mg of estrogen are associated with one third reduced risk when compared with proportion containing 50 mg.

Trauma :

Trauma is the leading cause of cerebro vascular mortality in the developing and developed countries. Blunt or penetrating trauma may result in cervico cephalic arterial dissection, arterial thrombosis, arterial rupture, pseudo aneurysm formation or development of AV fistula. Internal carotid artery thrombosis may also follow maxillary and mandibular angle fractures. Carotid artery hematoma may cause hematoma formation over the lateral neck, retinal or hemispheric ischemia and a Horner syndrome. Arteriography is indicated in most instances. There after surgical repair on angioplasty may be needed. (37)

Hypercoagulable states :

Primary hypercoagulable states are Antithrombin deficiency, protein C deficiency, protein S deficiency,Factor V leiden mutation(38), Activated protein C resistance, Prothrombin G 20210

mutation(39). Hypo or dysfibrinogenemia, hypoplasminogen, Lupus anticoagulant and anticardiolipin antibodies.

Secondary hypercoagulable states are malignancy, pregnancy, puerperium, oral contraceptive use, ovarian hyper stimulation syndrome, nephrotic syndrome, polycythemia rubra vera, paroxysmal nocturnal hemoglobinuria, heparin induced thrombocytopenia, homocystinuria, sickle cell disease, thrombotic thrombocytopenic purpura.

Vascular Anatomy of brain

The brain is perfused by the carotid and vertebral arteries. The carotid and its branches are referred to as anterior circulation and the vertebrobasilar as posterior circulation.

The right common carotid artery originates from the bifurcation of the innominate artery, whereas the left originates from the aortic arch. The internal carotid arteries arise from the common carotid artery usually at the level of upper border of thyroid cartilage at C4 vertebrae. They give no branches in the neck and face enter the cranium through the carotid canal. Internal carotid artery ends by dividing into middle

and anterior cerebral arteries after giving off the ophthalmic, superior hypophyseal, posterior communicating and anterior choroidal arteries.

Middle cerebral artery is the largest branch of the ICA and supplies entire lateral surface of the brain except frontal pole, superior and extreme posterior rim of convex surface and medial cortical surfaces. Upper division usually gives rise to lateral orbitofrontal, ascending frontal, prerolandic, rolandic and anterior parietal branches whereas lower division comprises anterior, middle, posterior temporal branches, posterior parietal and angular arteries. It supplies lateral surface of frontal, parietal, temporal areas, extreme capsule, claustrum putamen, globus pallidus, head and body of caudate nucleus, superior portions of internal capsule.

Anterior cerebral artery begins as a medial branch of internal carotid artery supplies medial and orbital surface of frontal lobe especially paracentral lobule. One of the largest branch is recurrent art of Huebnber which supplies the head of caudate nucleus.

Vertebral arteries arise from the subclavian artery courses through the transverse foramina, pierces the dura, and enter the cranial cavity to join the contralateral vertebral artery. Anterior and posterior

spinal arteries and the posterior inferior cerebellar artery which supplies the inferior surface of the cerebellum arises from the distal segment of vertebral artery. The lateral medulla is the site of injury in Wallenberg syndrome is supplied by multiple perforating branches of PICA, or medullary branches from the vertebral artery.

Basilar artery formed at the pontomedullary junction in the midline by union of 2 vertebral arteries. Anterior inferior cerebellar and superior cerebellar arteries perfuse the ventrolateral aspect of cerebellum and labyrinthine artery arises from basilar arteries supplies cochlea, labrynth, part of facial nerve. Basilar artery divides into two posterior cerebral arteries on either side. Thalamoperforating, thalamo geniculate, turberothalamic perforation arteries arises from posterior cerebral art and supplies hypothalamus, dorsolateral midbrain, lateral geniculate thalamus, PCA directly supplies medial and inferior surface of occipital and temporal lobe.

STROKE TYPES AND SUBTYPES

Types :

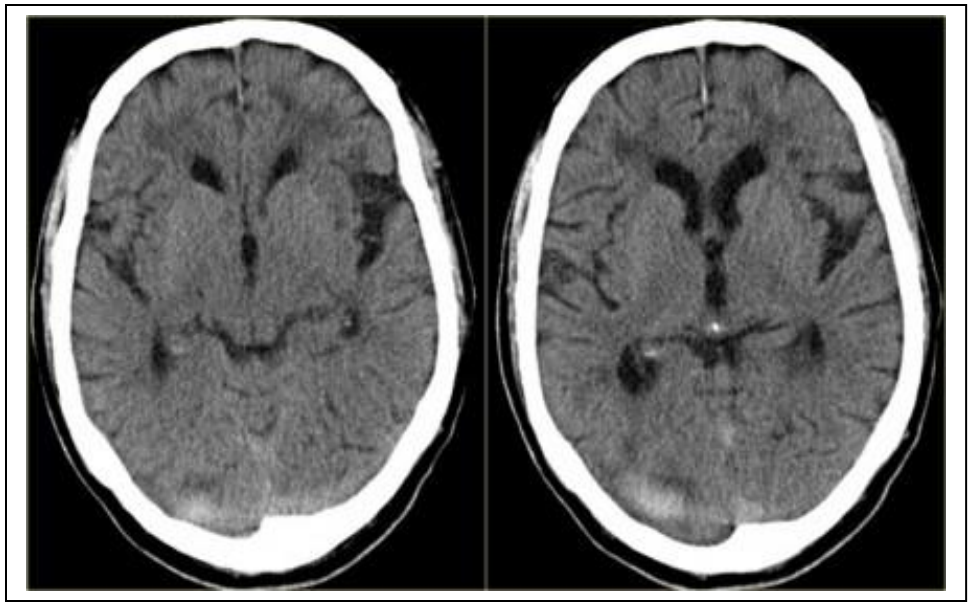
According to American Heart Association (AHA) Stroke is majorly classified into ischemic stroke and hemorrhagic stroke. Ischemic stroke accounts for 85% of all stroke remaining 15% strokes are due to hemorrhage. Ischemic strokes may be due to embolic or thrombotic. Thrombosis may occur in larger vessels or it may involve smaller vessels in lacunar stroke and may involve venous sinuses in cerebral venous thrombosis due to various etiology.

Ischemic strokes :

Thrombotic stroke :

It usually occurs in elder individual. The onset is gradual. Stuttering or step wise progression usually occurs. It often occurs during sleep or in early morning. So that patient awakens with deficit. A history of transient ischemic attack can be elicited in 50% of cases. This type of stroke is commonly though to result from insitu thrombosis in an intracranial vessels affected by atherosclerosis. Atheroma seems to be an almost inevitable accompaniment of ageing in developed and in developing countries. Atheroma is a multifocal

CEREBRAL VENOUS THROMBOSIS



LEFT PCA INFARCT

disease affecting both medium and large size arteries. Major sites of atheroma formation in anterior circulation are carotd bifurcation,carotid siphon, proximal MCA, and anterior communicating artery. Atheroma tend to affect both intra and extra cranial vessels and also affects small and medium sized vessels in the intracranially.

Male gender, older age, smoking, hypertension, diabetes, hypercholesterolemia are risk factors for carotid stroke.

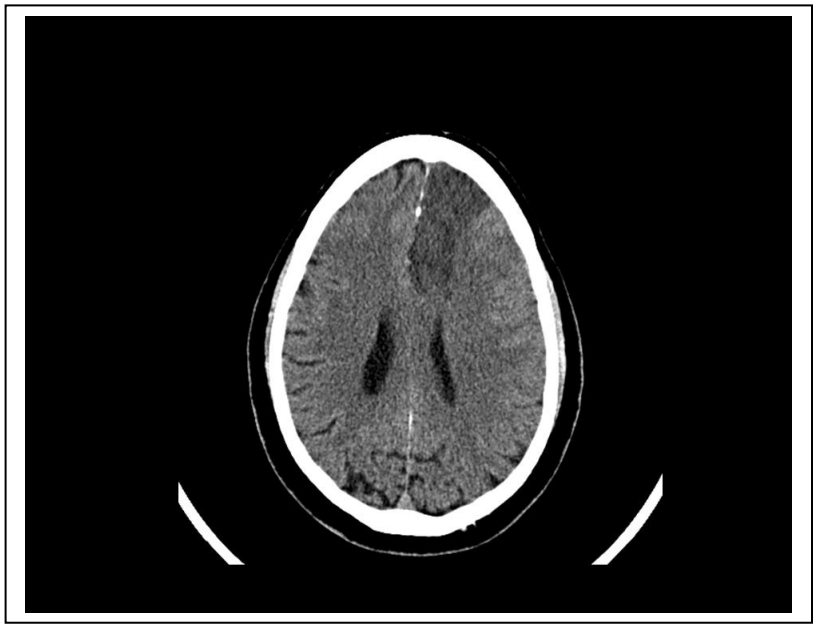
Symptomatic carotid disease implies that the patient has experienced a stroke or TIA with in the vascular distribution of the artery and it is associated with a greater risk of subsequent stroke than asymptomatic stenosis, in which the patient is symptom free and the stenosis is detected through screening.

Intracranial atherosclerosis produces stroke either by embolic mechanism or by in situ thrombosis of a diseased vessel. It is more common in Asian patients. Recurrent stroke risk is 15% per year, similar to symptomatic untreated carotid atherosclerosis.

Embolic stroke :

It usually occurs in younger patients and has a sudden onset often during usual daily activity. The deficit is maximal at the onset often

LEFT ACA INFARCT



RIGHT MCA INFARCT

improvement shortly afterwards as the embolus break up and travel further into more distal branches of the affected artery. Cardiac disease is the most common cause of embolism. Emboli may form over the atrial and ventricular wall or the left heart valves.

These thrombi then detach and embolize into the arterial circulation. Emboli from the heart more often lodge in MCA. Rarely ACA and PCA involved. Most common significant cause of cardioembolic stroke in India is atrial fibrillation more commonly in the setting of RHD. In developed countries it is non valvular atrial fibrillation. Other causes are myocardial infarction, prosthetic valves, cardiomyopathy, tachyarrythmias.

Patients with atrial fibrillation have on average annual risk of stroke 5%. The risk of stroke can be estimated by calculating the CHADS2 score.

Paradoxical embolisation occurs when venous thrombi migrate to arterial circulation via PFO and ASA.

Thrombus formation on atherosclerotic plaque may embolize to intracranial arteries producing an artery to artery embolic stroke.

Embolism of cardiac origin accounts for approximately 15 to 20% of all ischemic strokes. These cardiac emboli composed of platelet, fibrin, calcium, micro organism. The most common cause for cerebral embolism in younger individual is atrial fibrillation accounting for one half to two third of emboli of cardiac origin.

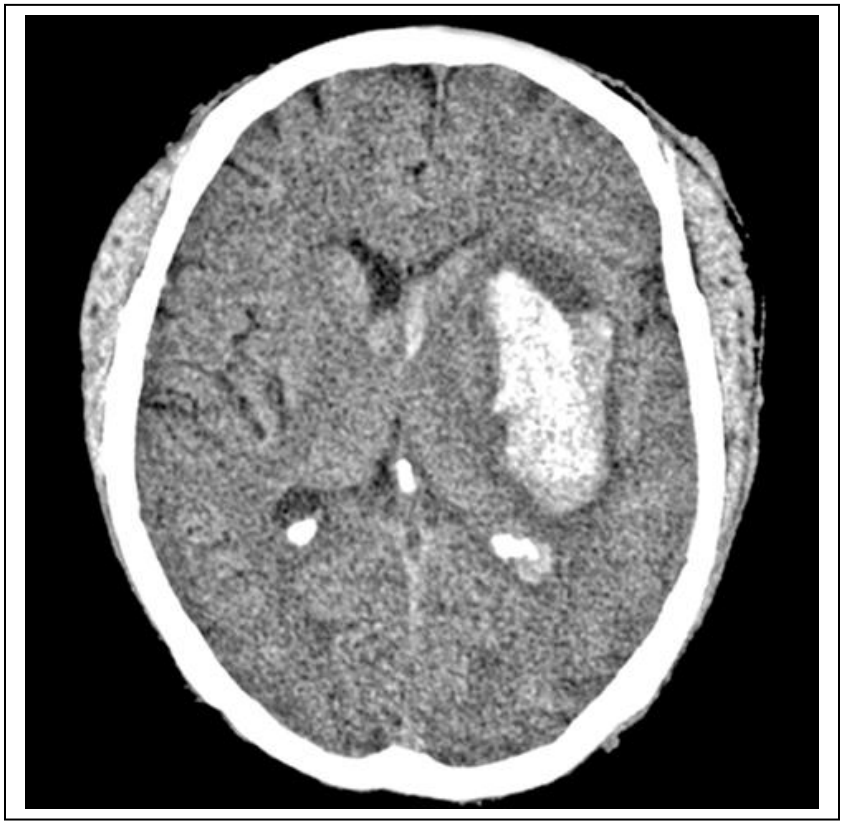
Congenital heart disease is probably most common cardiac disorder causing ischemic stroke in children. Those with a high hematocrit are more likely to experience cerebral venous thrombosis.

Acute MI accounts for 1% cadioembolic stroke. Left ventricle thrombi are commonly associated with recent anterior wall transmural MI. A decreased ejection fraction is an independent risk factor for stroke following MI(40). Left atrial spontaneous echocardiac graphic contrast and decreased atrial ejection force increase stroke risk (41). The risk for thromboembolism is also higher among patients in chronic atrial flutter(42). Aortic valve calcification with or without stenosis is also a major risk factor(43).

Hemorrhagic stroke :

Intracerebral hemorrhage accounts for approximately 10% of all strokes(44). It has high mortality when compared to ischemic strokes.

HAEMORRHAGIC STROKE



Mortality due to hematoma size and location. Over all mortality for this hemorrhagic stroke varies between 25 to 60%. Intracerebral hemorrhage is a major public health problem especially in populations at high risk Such as young and middle aged people. Genetic factors such as the possession of the E2 and EA alleles of the apolipoprotein E play an important role in the occurrence of certain forms of ICH such as lobar hemorrhage(45).

The main cause of ICH is hypertension. The primary role of hypertension in ICH is supported by high frequency of hypertension. The role of hypertension as a cause of ICH is most relavant for the non lobar locations which may be due to hypertension in approximately 50% cases(46). In both hypertensive and non hypertensive patients circadian rhythm of ICH onset which peaks at 8AM and 8 pm coincide with the physiological daily peaks of blood pressure pointing to the importance of blood pressure rises in the pathogenesis of ICH(47).

ICH caused by AVM or cavenous angioma are frequently located in the subcortical white matter. The hematoma is generally smaller and symptoms develop more slowly than hypertensive hemorrhage.

Bleeding disorders caused by abnormalities of coagulation are rare cause of ICH. Hemophilia caused by factor 8 deficiency also lead to ICH . Cerebral amyloid angiopathy may play a causal role in the ICH that occur in patients treated with anticoagulants(48).

Fibrinolytic agents like streptokinase tissue plasminogen activator can also cause ICH. rtPA used for the treatment of acute ischemic stroke was complicated with ICH in 6% of cases(49). These hemorrhage occur at the site of the proceeding cerebral infarct are generally large and carry a dismal prognosis(50).

Cerebral amyloid angiopathy is characterized by selective deposition of amyloid in cerebral vessels. It steadily increases with age. Less common in age < 55 years. Superficial location of the affected vessels in the cortex and Leptomeninges is responsible for lobar location in ICH.

Cocaine has become the most common sympathomimetic agent associated with ICH. Both ICH and SAH can occur with in short periods. ICH due to vasoconstriction drug induced cerebral vasculitis and coexisting AVM(51). Amphetamine can also cause ICH(52).

Vasculitides like PolyArteritis Nodosa is known to present with ICH.

Decongestant and appetite suppressant phenylpropolamine has been associated with ICH in young patients especially in women.

OUTCOME and FOLLOW UP

Modified RANKIN scale and Modified BARTHEL index of activities of daily living were used for assessing the improvement in motor weakness and unsteadiness. Modified Rankin scale and Barthel index at the time of admission and at 90 days were compared and analysed.

MODIFIED RANKIN SCALE

SCORE	DESCRIPTION
0	No symptoms at all
1	No significant disability, able to carry out all usual activities and duties
2	Slight disability, unable to carry out previous activities, able to carry out works with out assistance
3	Moderate disability, requiring some help, but able to walk without assistance
4	Moderately severe disability, unable to walk without assistance, unable to attend own bodily needs without assistance.
5	Severe disability, bedridden, incontinent, requiring constant nursing care and attention .
6	Dead .

MODIFIED BARTHEL INDEX(MBI)

Item	Unable to Perform task	Substantial Help need	Moderate Help need	Minimal Help need	Fully independent
Personal hygiene	0	1	3	4	5
Bathing self	0	1	3	4	5
Feeding	0	2	5	8	10
Toilet	0	2	5	8	10
Stair climbing	0	2	5	8	10
Dressing	0	2	5	8	10
Bowel control	0	2	5	8	10
Bladder control	0	2	5	8	10
Ambulation	0	3	8	12	15
Wheel chair	0	1	3	4	5
Chair/bed transfer	0	3	8	12	15

Modified Barthel Index(MBI) scoring system

MBI total score	Dependency level
0-24	Total
25-49	Severe
50-74	Moderate
75-90	Mild
91-99	Minimal

MATERIALS AND METHODS

The present study had been conducted at Government Rajaji Hospital, Madurai Medical College, Madurai during the period between March 2013 and February 2014. I have obtained written consent from the patient, after explaining about the study in detail. I got permission from ethical committee for this study from Madurai Medical College. The inpatients admitted in Neurology and Medical ward during one year period between March 2013 to February 2014 were taken up for the study. The clinical details obtained from ward register, case sheets and patient interviews with the help of the previously prepared proforma. I have taken details on demographic profile, stroke history, and treatment history by interviewing the patient and from the case history with the help of standard questionnaire. Associated symptoms, risk factors for stroke were noted in proforma. The diary will be scrutinized monthly during the period of the survey.

Study design :

Prospective cross sectional observation study

Period of study :

March 2013 to February 2014

Study population :

100 stroke patients who admitted in neurology and medical wards, in Govt. Rajaji Hospital, Madurai Medical College, Madurai between March 2013 to February 2014.

Inclusion criteria :

1. Patients admitted in wards, Govt. Rajaji Hospital, Madurai
2. Age < 45 years
3. Not only arterial stroke but also venous infarct (CVT) were included in this study.

Exclusion criteria :

1. Those paediatric age group patients and patients above 45 years are strictly excluded.
2. Patients who developed post ictal weakness following seizures were excluded.
3. Patients with weakness due to infections (TB, Neuro cysticercosis, Toxoplasmosis), Tumour like meningioma were excluded.

4. Weakness due to trauma excluded.
5. Those with focal neurological deficits due to HIE, Attempted hanging, Poisoning were excluded
6. Patients refusal to participant in the study were excluded.

Calculation of sample size

100 patients of young stroke admitted in neurology and medical wards were included and analysed between March 2013 to February 2014. It is only a prospective study.

Statistical Analysis :

The information collected regarding all the selected cases were recorded in a master chart. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012). Using this software, range, frequencies, percentage, mean, standard deviation and 'p' value were calculated through One way ANOVA, Chi square, Pearson and Spearman Correlation test and P value of < 0.05 was taken as significant.

OBSERVATION

Table – 1

Age Distribution

Age in years	No. of cases	Percentage
15 – 25	10	10
25 – 35	27	27
36 - 45	63	63
Total	100	100

In this study, 63 cases in 36 - 45 years age group is higher when compare to other age groups. 'p' value is <0.001 significant.

AGE DISTRIBUTION

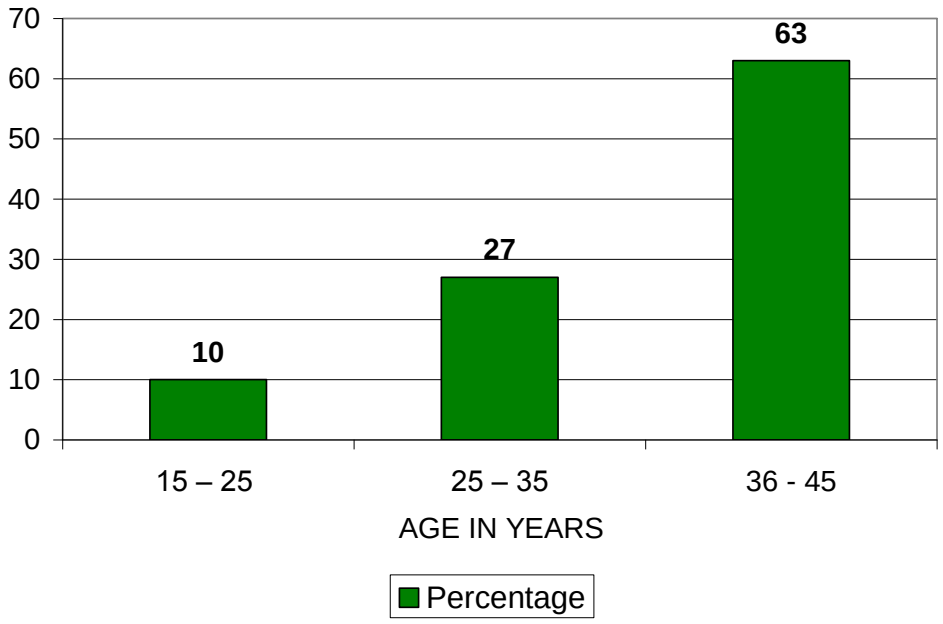


Table – 2

Sex Distribution

Sex	No. of cases	Percentage
Male	63	63
Female	37	37
Total	100	100

In this study males are affected more than females and P value is statistically significant 0.045.

SEX DISTRIBUTION

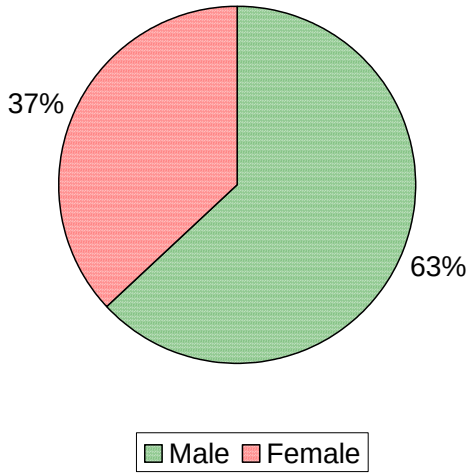


Table – 3

Stroke major types

Types	No. of cases	Percentage
Ischemic	84	84
Hemorrhagic	16	16
Total	100	100

In this study ischemic stroke is significantly higher than hemorrhagic stroke and ‘p’ value is <0.001 significant.

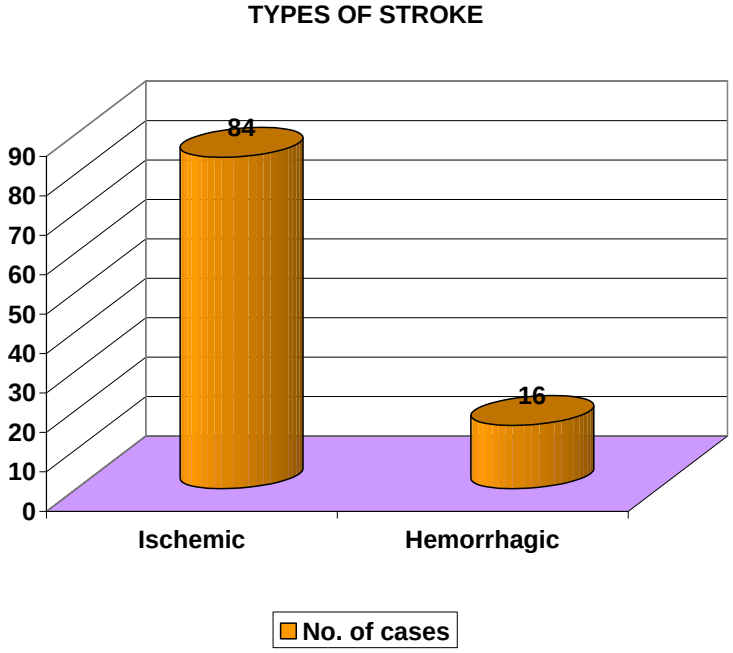

TYPES OF STROKE
84
16
90
80
70
60
50
40
30
20
10
0
Ischemic
Hemorrhagic
No. of cases

Table – 4

Ischemic Stroke sub types (TOAST)

(Total cases 84)

Types	No. of cases	Percentage
Large vessel atherosclerosis	36	42.9
Cardiac embolism	28	33.3
Small vessel atherosclerosis	8	9.5
CVT	12	14.3

In this study thrombotic strokes are more common than cardioembolic strokes and ‘p’ value is <0.001 significant.

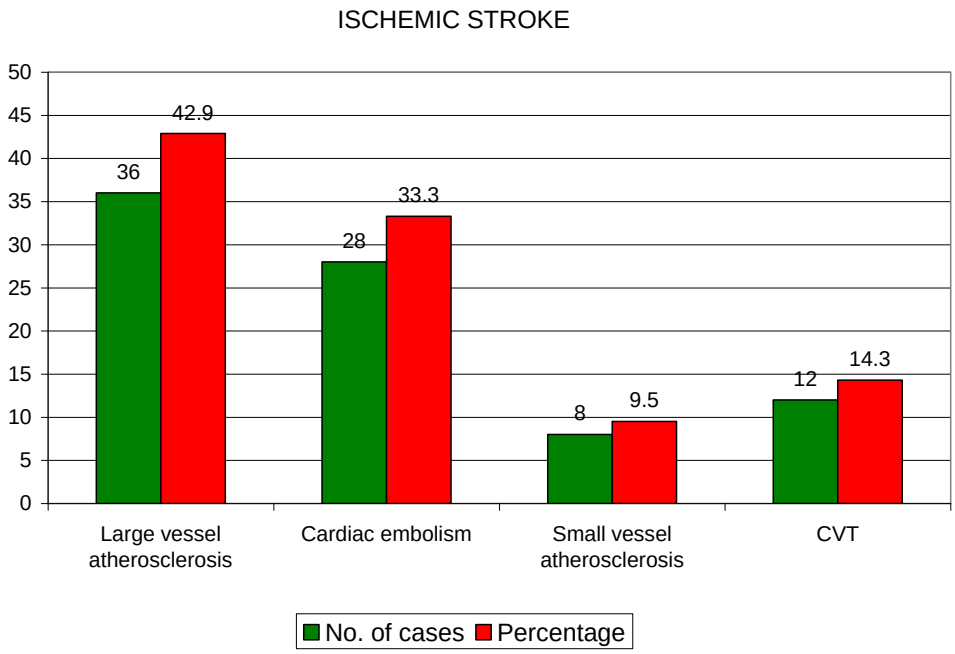

ISCHEMIC STROKE
50
45
40
35
30
25
20
15
10
5
0
36
42.9
28
33.3
8
9.5
12
14.3
Large vessel atherosclerosis
Cardiac embolism
Small vessel atherosclerosis
CVT
No. of cases
Percentage

Table – 5

Arterial Territory distribution

(Total arterial stroke cases 80)

Artery	Total cases	Male	Female	Percentage
Anterior circulation stroke				
Anterior cerebral artery	11	8	3	13.8
Middle cerebral artery	56	38	18	70.0
Posterior circulation stroke	13	9	4	16.2

In this study MCA more commonly involved than ACA and PCA and 'p' value is <0.001 significant.

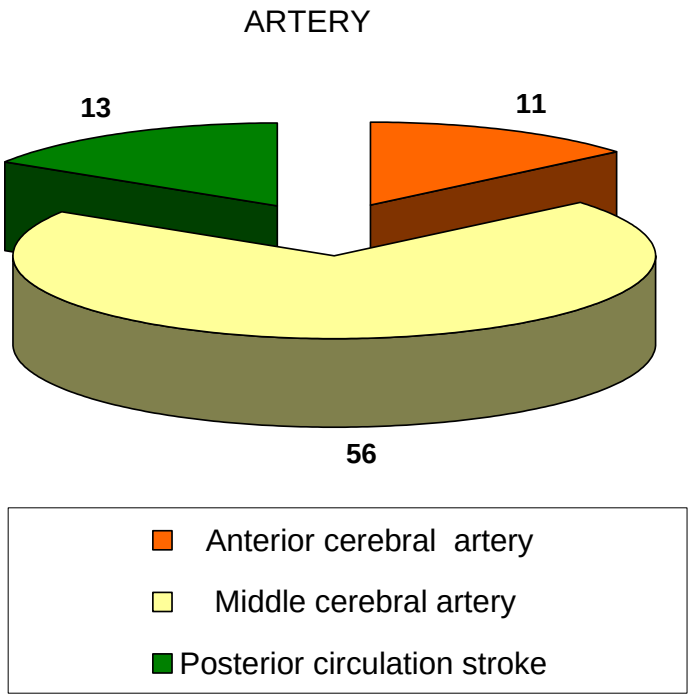

ARTERY
13
11
56
Anterior cerebral artery
Middle cerebral artery
Posterior circulation stroke

Table – 6

Aphasia

(Total aphasic cases 38)

Types	No. of cases	Percentage
Pure aphasia	10	26.3
Aphasia with weakness	28	73.7

Table – 7

Types of Aphasia

(Total aphasic cases 38)

Aphasia	No. of cases	Percentage
Motor	24	63.2
Sensory	6	15.7
Global	8	21.1
Other types	0	0

In this study motor aphasia more commonly involved than other aphasias and ‘p’ value is <0.001 significant.

TYPES OF APHASIA

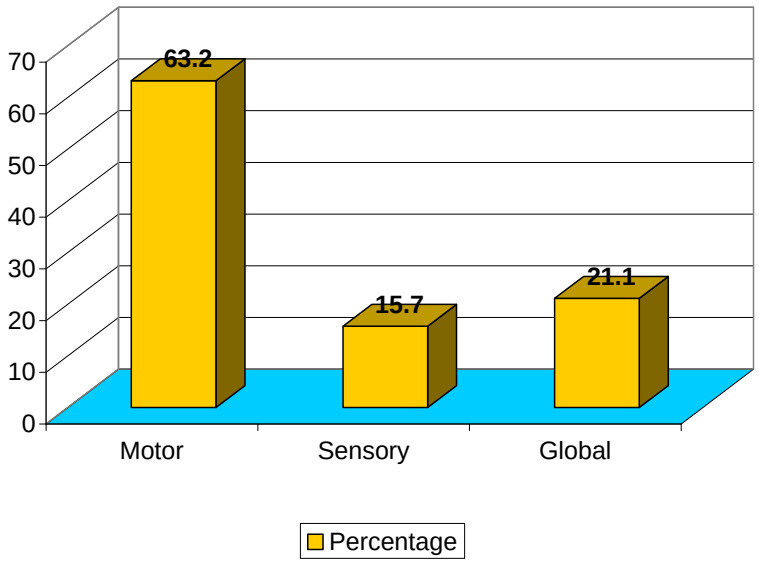


Table – 8

Various presentations on admission

(Total cases 100)

S.No.	Symptoms on admission	Total	Male	Female
1.	Weakness	54	33	21
2.	LOC	15	10	5
3.	Unsteadiness	13	9	4
4.	Speech disturbances	10	7	3
5.	Headache	4	3	1
6	Lower CN palsy	2	2	0
7	Sensory symptom	1	0	1
8	Seizures	1	1	0

In this study weakness is more common than other presentations and 'p' value is <0.001 significant.

PRESENTATION

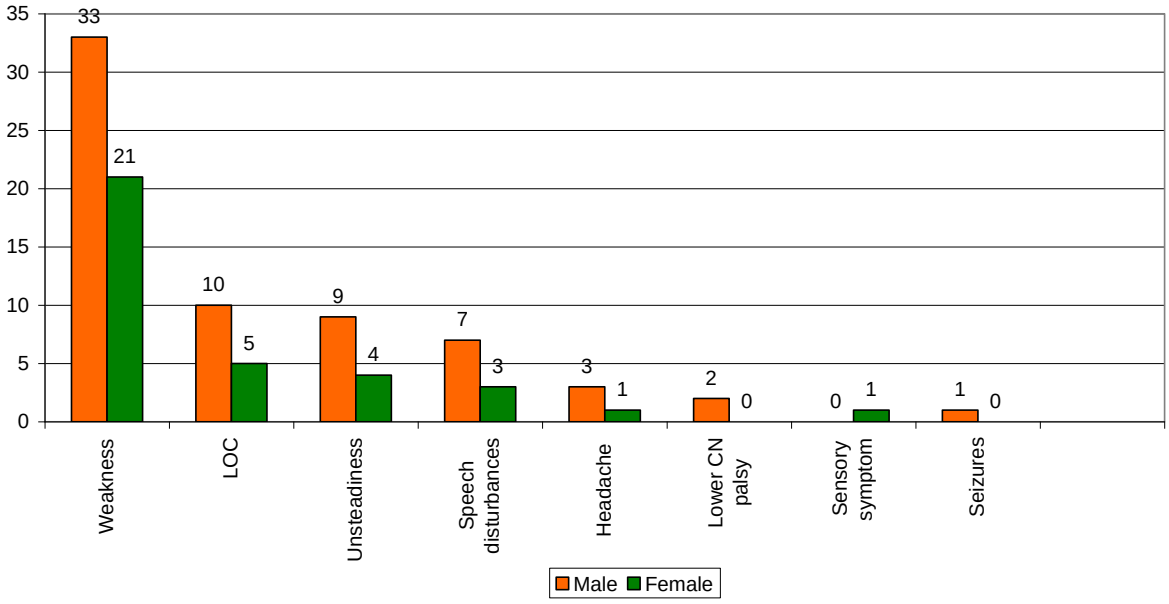


Table – 9

Pattern of weakness

(Total cases 82)

S.No.	Rt sided weakness		Lt sided weakness	
Total	53		29	
Sex	Male	Female	Male	Female
	33	20	22	7
Percentage	64.6		35.4	

In this study right sided weakness is more common than left sided weakness and‘p’ value is 0.042 Significant.

Table – 9 a

Pattern of weakness

(Total cases 82)

Weakness	No.of cases	Percentage
Hemiplegia (Power < 3)	31	37.9
Hemiparesis (Power > 3)	51	62.1

Table – 10

Risk factor for ischemic stroke

Risk factors	Total	%	Male	%	Female	%
Diabetes	25	29.7	19	22.6	6	7.1
Hypertension	26	31	19	22.6	7	8.4
Smoking	43	51	43	51	0	0
Alcohol	38	45.2	38	45.2	0	0
Dyslipidemia	33	39.2	20	23.8	13	15.4
CAD	18	21.4	12	14.2	6	7.2
Valvular lesion	26	31	8	9.5	18	21.5
Rheumatoid factor	3	3.6	0	0	3	3.6
ANA	1	1.2	0	0	1	1.2
Homocysteinemia	1	1.2	1	1.2	0	0
APLA	1	1.2	0	0	1	1.2
Hypercoagulable status	4	4.8	1	1.2	3	3.6

In this study, diabetes, hypertension, smoking, alcohol, dyslipidemia are the most common risk factors for ischemic stroke. and its ‘p’ value is <0.001 significant.

Table – 11

Hemorrhagic stroke

(Total cases 16)

		Hypertension								Bleeding diathesis	
Idiopathic		Renal parenchymal disease		RAS		Adrenal tumour		Coarctation of aorta		Hemophilia	
3		7		2		2		1		1	
M	F	M	F	M	F	M	F	M	F	M	F
2	1	5	2	0	2	2	0	1	0	1	0

Table – 12

Cardio embolic stroke (Total cases 28)

Lesion	Total cases	Male	%	Female	%
RMD MS/AF	12	5	17.9	7	25.0
RHD MS/MR/AF	8	1	3.6	7	25.0
CHD ASD	6	2	7.i4	4	14.28
Cardio myopathy	2	1	3.6	1	3.6

Table – 12 A

Atrial fibrillation

	No.of cases	Percentage
AF positive cases	24	85.7
AF negative cases	4	14.3

In this study Atrial fibrillation is a potential risk factor for cardio embolic stroke and 'p' value is 0.003 significant.

ATRIAL FIBRILLATION

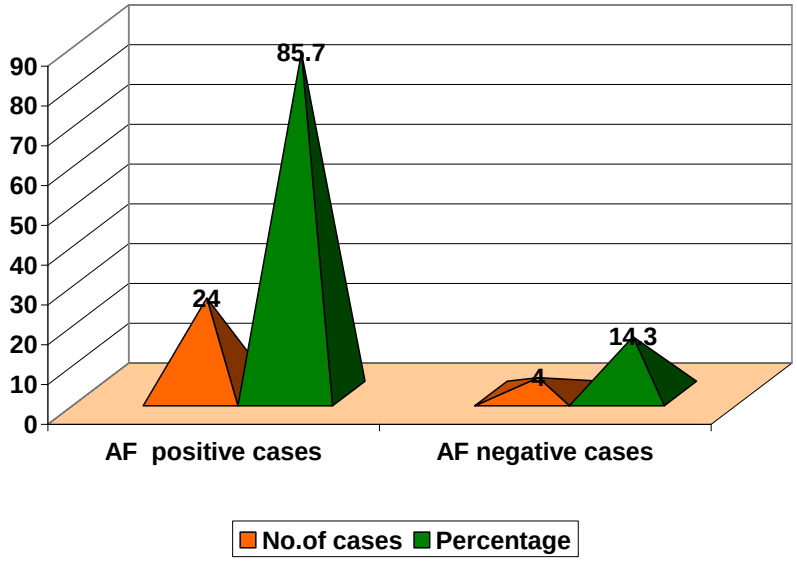


Table – 13

Thrombotic strokes

(Total thrombotic cases 56)

(Total Carotid Doppler +ve cases 40)

	No. of cases	Percentage
Carotid Doppler positive cases	30	75
Carotid Doppler negative cases	10	25

In this study carotid Doppler positivity indicative of atherosclerosis is a major risk factor for thrombotic strokes and 'p' value is <0.001 significant.

THROMBOTIC STROKE

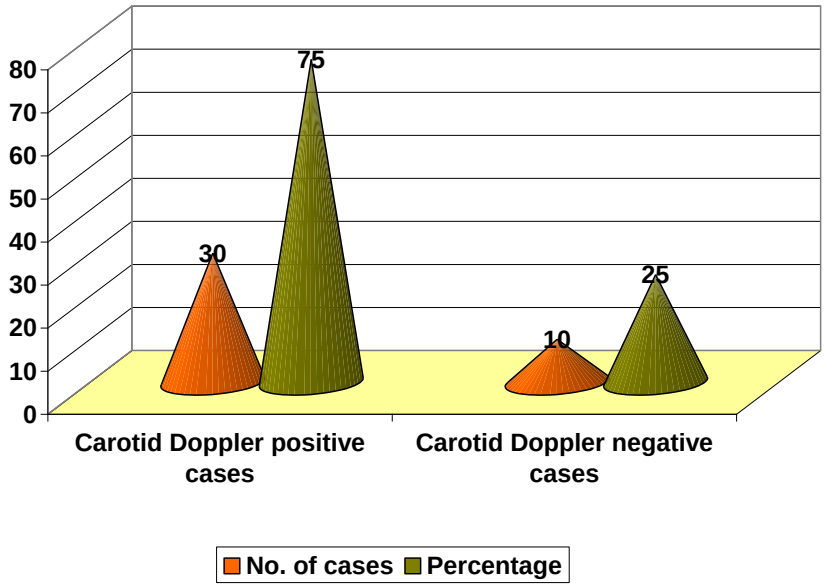


Table – 14

BMI

BMI	Male	Female	Total
Normal < 25kg/m2	10	8	18
Overweight 25 to 29.9 kg/m2	20	7	27
Obesity > 30 kg/m2	38	17	55
Total	61	39	100

In this study Obesity is more common compared to normal individuals and ‘p’ value is <0.001 significant.

Table – 15

Outcome - Weakness

S.NO	MRS SCORE	Total no of cases on admission	Follow up at 90 days
1	0	0	0
2	1	8	41
3	2	15	35
4	3	25	12
5	4	32	5
6	5	12	3
7	6	8	4

Table 15 a

Outcome -weakness

Barthel index Scoring	Dependency level	Total cases on admission	At 90 days follow up
0-24	Total	13	8
25-49	Severe	18	12
50-74	Moderate	42	23
75-90	Mild	20	52
91-99	minimal	7	5

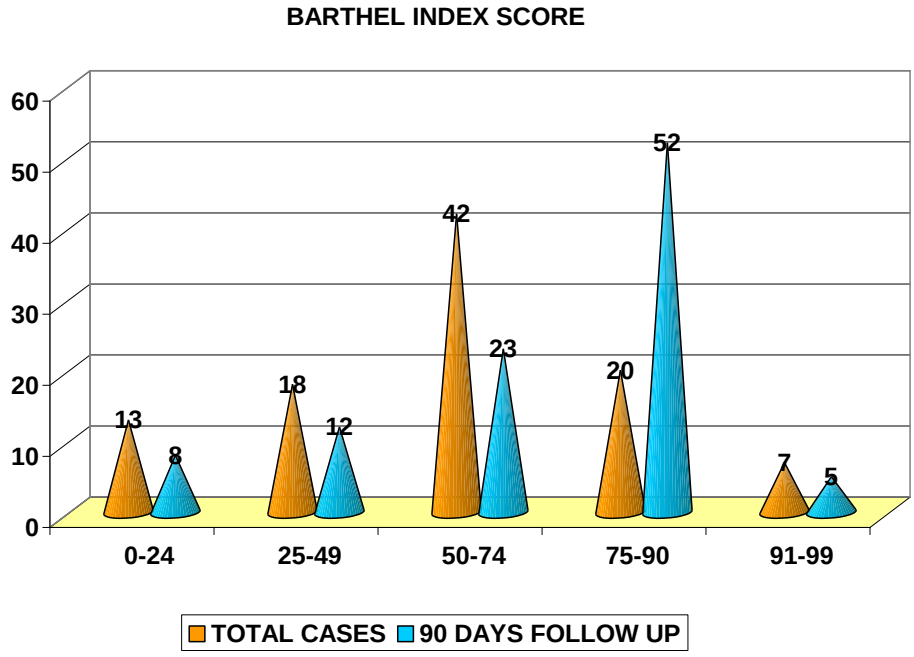

BARTHEL INDEX SCORE
60
50
40
30
20
10
0
13
8
18
12
42
23
20
52
7
5
0-24
25-49
50-74
75-90
91-99
TOTAL CASES
90 DAYS FOLLOW UP

Table – 16

Outcome in Speech disturbances

Speech disturbances	Total cases	%				
			Improved	%	Not improved	%
Motor aphasia	24	63.2	21	87.5	3	12.5
Sensory aphasia	6	15.7	5	83.3	1	16.7
Global aphasia	8	21.1	6	75.0	2	25.0

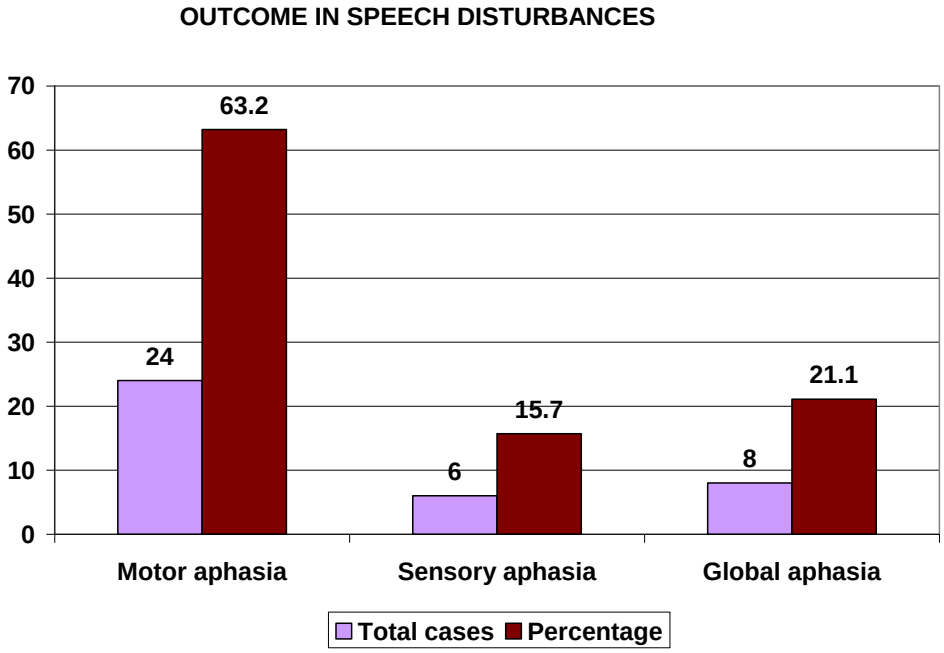

OUTCOME IN SPEECH DISTURBANCES
70
60
50
40
30
20
10
0
63.2
24
15.7
6
21.1
8
Motor aphasia
Sensory aphasia
Global aphasia
Total cases
Percentage

Table – 17

Outcome in Unsteadiness

(Total cases of unsteadiness 13)

	No.of cases	Percentage
Improved	3	23.1
Not improved	10	76.9

In this study unsteadiness not improved significantly and p value is significant <0.001

Table – 18

Outcome -Death

(Total death cases 12)

	No.of cases	Percentage
Immediate	8	66.6
Late	4	33.3

RESULTS AND ANALYSIS

In the present study which was conducted over 1 year observation period, totally 100 patients completed the study and they were analysed. Their mean age was 38.9 yrs and 61 of them were male and 39 were female.

Table 1 showed Age distribution in young stroke patients. Young stroke (under 45 years) is more common in the age group between 35 to 45 years. Total no. of cases is in 35 to 45 yrs is 63. The percentage is 63%. Total no. of cases in the age group 25 to 35 yrs is 27. The percentage is 27%. Total no. of cases in the age group 15 to 25 yrs is 10. The percentage is 10%. Our study correlated with Mayo study. Atherosclerosis is the chief cause of cerebral ischemia. As atherosclerosis is an age related process incidence of stroke is probably less common in age group 25 to 35 yrs when compared to 35 to 45 yrs.

Table 2 showed sex distribution in young stroke. Males constitute 63% of young stroke. Females constitute 37% of young stroke in our study. Out study correlate with the Chan MT Morris et al study. Male gender prone for stroke than females.

Table 3 showed major stroke types. Ischemic stroke more common than hemorrhagic stroke. Ischemic stroke contribute 84%, hemorrhagic stroke contributes 16% cases in our study.

Table 4 showed ischemic stroke subtypes in young stroke. Thrombotic stroke common than cardio embolic stroke in our study. Thrombotic stroke contributes 53%, Cardio embolic stroke contributes 33%, Cerebral venous thrombosis contributes 14% cases. Our study correlates with Kwon,Varona, and Carolei et al study.

Comparison study

Study	%Athero thrombotic	%Cardioembolic	%Others
kwon et al	52	30	27
Carolei et al	48	32	8
Varona et al	48	30	12
Our study	53	33	19

Table 5 showed Arterial territory in young stroke. In our study anterior circulation strokes commonly involved than posterior circulation. In the anterior circulation middle cerebral artery more

commonly involved than anterior cerebral artery. MCA stroke contributes 70%, ACA stroke contributes 14% and Posterior circulation stroke contributes 16%.

Table 6 showed speech disturbances in young stroke. Aphasia seen in 38 cases accounts for 38% of total young stroke cases. Of these pure aphasia seen in 10 cases(26%), aphasia with weakness seen in 28 cases(74%).

Table 7 showed types of aphasia in 38 patients of young stroke. Of all the aphasia motor aphasia more commonly involved than other aphasia. Motor aphasia contributes 63%. Sensory aphasia contributes 16%, global aphasia contributes 21 % of total cases.

Table 8 showed the different mode of presentation in young stroke. Motor weakness more common than others. Weakness contributes 54%, Unsteadiness contributes 13%cases, Unconsciousness contributes 15% cases, Pure Language disturbances seen in 9% cases, Headache alone contributes 4% of cases, Seizures alone contributes1% cases, lower cranial palsies in 2% of cases, sensory symptom in 1% of case.

Table 9 showed the different pattern of motor weakness in young stroke among the motor weakness. Right sided weakness (65%) most commonly present than left(35%).

Table 9a showed the grading of weakness. Hemiparesis (power>3) most commonly presented than hemiplegia (power<3). Hemiparesis seen in 62% of cases, Hemiplegia seen in 38% of cases.

Table 10 showed risk factor analysis. Males are most commonly affected than females. Males accounts for 63 % of cases, Smoking, and Alcohol is the most common risk factors in male. Smoking alone seen in 48% cases, alcoholism alone in 38%, both smoking and alcoholism seen in 68% of cases.

Hypertension, Dyslipidemia, Coronary artery Disease, valvular lesions are the most common risk factors for stroke. Of these Hypertension, Dyslipidemia and Coronary artery disesae are common in males whereas Valvular lesions are common in females in our study.

Positive ANA and positive Rheumatoid factors, and positive APLA are the risk factors present only in females in our study. ANA

positivity seen in 1 cases, Rheumatoid factor positivity in 3 cases, APLA positivity seen in one case.

Homocystinemia seen in 1 case responsible for thrombotic stroke in male patient.

Hypercoagulable status was positive in 4 cases, and causes CVT in 4 patients. Of these 4 patients one patient was male others 3 were female. Protein C deficiency was seen in one male patient. Two female patients had protein C deficiency and another female patient had protein S deficiency. The other remaining 8 patients were female all are in postpartum state

Table 11 showed risk factors for Hemorrhagic stroke. Hypertension is the most common cause for hemorrhagic stroke. Of the 16 hemorrhagic stroke cases 15 cases were hypertension induced and one cases of hemorrhagic stroke was due to Hemophilia induced. Hemorrhagic stroke seen in 11 males cases, and in 5 female cases. While evaluation of secondary hypertension chronic renal failure seen in 7 cases, Renal artery stenosis seen in 2 cases, Idiopathic hypertension seen in 3 cases, Adrenal tumour seen in 2 cases Coarctation of aorta seen in one case.

Table 12 cardiac cause as a risk for cardiembolic stroke seen in 28 cases. Of these Valvular lesions seen in 26 cases. 2 cases presented with cardiomyopathy. Of the 26 valvular cases RHD accounts for 20 cases. In all these RHD cases atrial fibrillation seen. ASD seen in 6 cases. Atrial fibrillation present in 5 cases. Hence our study conclude that presence of atrial fibrillation in heart disease increases the risk of cardio embolic stroke.

Table 12A showed significant positivity of Atrial fibrillation in cardio embolic strokes

Table 13 showed out of 56 Thrombotic stroke cases 40cases showed positive carotid Doppler atherosclerotic plaque lesions. Of these 30cases were male and 10 cases were female.

Table 14 showed analysis of BMI in 100 cases, 55 cases were obese (BMI > 30), over weight were 27 cases (BMI 25-30), Normal (BMI < 25) seen in 18 cases. In our study > 50% of our young stroke patients were comes under metabolic syndrome and responsible for both stroke and CAD.

Table 15 showed outcome in weakness at 90 days, on admission more than 50% of cases were clustered in MRS scoring of 3-4.after 90 days follow up more than 50%of cases were clustered in MRS grading of 1-2 showing significant improvement in motor weakness.

Table15a showed otcome in weakness.On admission most of the were clustered in modified BARTHAL index SCORING of moderate to severe stage (25-74 score). After 90 days follow up most of the cases were clustered in mild MBI staging (score 75-96)

Table 16 showed during follow at 180 days speech disturbances were assessed. Motor aphasia was improved in 21 cases (87.5%) and persisted in 3 cases(12.5%). Sensory aphasia improved to nominal aphasia in 5 cases (83.3%) persistence of sensory aphasia seen in 1 cases (16.7%). Global aphasia improved to motor aphasia in 6 cases (75%) and it was persisted in 2 cases (25%).

Table 17 showed out come in unsteadiness. Unsteadiness was improved only in 3 cases(23.1%). Persistence of unsteadiness was seen in 10 cases(76%) during 1 year follow up.

Table 18 showed the number of cases death in our study during follow up. Total number of death was 12 cases. Immediate death seen in 8 cases(66.6%) and late death seen in 4 (33.3%)cases.

DISCUSSION

The stroke is the leading cause of death and morbidity world wide. A higher proportion of younger individuals suffer from stroke among developing countries as compared with developed countries.

By knowing the prevalence of risk factors in both stroke and stroke subtypes we can improve the primary and secondary preventive strategies.

Stroke in the young requires a different approach to investigations and management than stroke in the elderly given differences in the relative frequencies of possible underlying cause.

In the following discussion I explore the different etiological risk factors in young stroke, their clinical presentation and their outcome in further follow up.

Etiopathogenic risk factors:

According to Carolei et al(53), Kwon et al(54), Varona et al(55) Of all subtypes of stroke in young adults found that thrombotic stroke was the most common subtype followed by embolic and hemorrhagic.

Our study correlates with above study. In our study thrombotic stroke seen in 44 cases (44%) cardio embolic stroke in 28 cases (28%) hemorrhagic strokes in 16 cases (16%), CVT in 12 cases (12%)

Overall there is males are most commonly affected in stroke. Studies(Nayak et al, Lipska et al) performed on ischemic stroke among the 15-45 age group from India also reported a male preponderance56,57

The proportion of cases in our study is higher in the age group between 35-45 yrs which is similar to findings reported by Nayak et al (56).

Presenting symptoms are similar to Chopra, Prabakar and Nayak et al study, In our study embolic stroke presented with profound loss of power in limbs(58,59).Mostly they occur in the afternoon session. Thrombotic stroke occurs mostly in the early morning while got up.

According to Mehindiratta MM et al Smoking, Alcoholism, Hypertension have been found to be significantly associated with ischemic stroke. Our study also correlates with this study. Smoking seen in 43%, Alcoholism seen in 38%, Hypertension seen in 28% of ischemic stroke cases.

Diabetes was not found to be a risk factor in Sweden and Taiwan but in out study (25%) Diabetes is a significantly risk factor. It corresponds to Lipska study.

Dyslipidemia (hyper cholesterolemia, hyper tryglyceredemia) known to be associated with ischemic stroke in young adults. Dyslipidemia significantly seen in 33 out of 52 ischemic stroke contributing atherosclerosis in our study. Our study correlates with the Arnold M et al study

Elevated homocysteine level seen in one case responsible for thrombotic stroke in male patient.

Antiphosphotolipid antibody syndrome seen in one case responsible for thrombotic stroke in female.

Vasculitis is also one of the cause of young stroke. Vasculitis causing stroke seen in 4 patients in our study. Of these Rheumatoid arthritis contributes 3 cases .SLE seen in 1 case (1.68%). All 4 cases were seen in females.

Hypercoagulable status is a rare cause of young stroke. In our study hypercoagulable status seen in 4 cases. All contributes CVT in

our study. Among 4 patients, 1 patient was male and remaining 3were females.

Clinical presentation :

Stroke may presented with various symptoms and signs. Like weakness, numbness, confusion, speech disturbances, slurring of speech, hemianopia, visual loss, headache, convulsion, unsteadiness giddness and unconsciousness.

Of the 100 cases of stroke Weakness (Loss of power) most commonly seen. Weakness seen in 82 cases. Of the weakness Right sided weakness is more common seen in 52 cases (63%). Lt sided weakness seen in 30 cases (37%).

Speech disturbances seen in 38 cases. Pure speech disturbances seen in 10 cases (26.3%)) speech disturbances with weakness in 28cases(73.7%). Of the various speech disturbances motor aphasia more common than other aphasias seen in 24 cases (63.2%) Sensory aphasia seen in 6 cases (15.7%) global aphasia seen in 8 cases (21.1%).

Headache is a common symptom in hemorrhagic stroke. In our study headache seen in 4 cases. All cases are hemorrhagic stroke.

Unsteadiness is a common symptoms in posterior circulation stroke. In our study Unsteadiness seen in 13 cases. All are due to posterior circulation stroke. Among these 7 patients also had lower cranial nerve palsy (9, 10 cranial nerves)

Unconsciousness seen in 15 cases in our study, of these 15 cases 11 cases were due to hemorrhagic stroke other 4 cases were due to massive infarct.

Convulsions are uncommon in stroke. It can occur in embolic stroke, hemorrhagic stroke and in cerebral venous thrombosis. In our study convulsions alone are the least symptom seen in one case due to embolic stroke. Convulsions with weakness usually seen in CVT. Our 4 patients had similar pattern all are due to CVT.

Obesity is one of the features of the metabolic syndrome responsible for stroke and CAD in both younger and older individuals. In our study obesity was common due to sedentary life style. Obesity in our study seen in > 50% (55 cases). Over weight individuals contributes 27% of young stroke cases.

RHD is the most common etiology for cardio embolic stroke in our study. RHD seen in 20 cases of all cardioembolic strokes (28

cases). ASD seen in 6 cases. Cardiomyopathy seen in 2 cases. Among the 28 cardio embolic cases Atrial fibrillation seen in 24 cases. Hence Atrial fibrillation is the potential risk factor for embolic stroke.

Hypertension is the major risk factor for hemorrhagic stroke. Among the 16 cases of hemorrhagic stroke hypertension was present in 15 cases. One case was due to bleeding diathesis (Hemophilia). Secondary causes of hypertension was assessed. Renal artery stenosis seen in 2 cases. Adrenal tumor seen in 2 cases. Coarctation of aorta seen in 1 case. Idiopathic hypertension seen in 2 cases. Chronic kidney disease seen in 7 cases.

Outcome :

During followup study over a period of 6 months to 1 year weakness, speech disturbances and unsteadiness were assessed.

Weakness and speech disturbances were improved significantly more than unsteadiness.Motor weakness were improved significantly according to Modified RANKIN scale and modified BARTHEL index.

Motor aphasia recovered to normal speech in 21 cases. Sensory aphasia recovered to nominal aphasia seen in 5 cases. Global aphasia

recovered to motor aphasia seen in 6 cases after intense speech therapy after a period of 6 month to 1 year..

Unsteadiness was not much improved when compared to motor weakness. In our study unsteadiness seen in 13 cases. Only 3 cases were improved partially and able to walk independently. Remaining 10 cases were not improved required assistance to walk.

No. of cases of death were 12 in our study. Immediate death seen in 8 cases. Of these 4 cases due to massive cerebral edema, 2 cases were due to associated systemic disease. 2 cases were due to electrolyte imbalances. 4 cases were died during follow up study. Of these 2 cases were due to aspiration pneumonia and respiratory failure. 2 cases were due to bedsores and septicemia.

SUMMARY AND CONCLUSION

1.Stroke under 45 yrs is common in the age group between 35-45 years.

2.Males are affected more than females in young stroke

3.Ischemic stroke more common than hemorrhagic stroke.

4.Thrombotic stroke more common than embolic stroke in ischemic strokes

5.Anterior circulation strokes more common than posterior circulation strokes.

6.MCA more commonly involved than ACA and PCA.

7.Motor weakness is the common presentation of young stroke followed by unsteadiness, unconsciousness and speech disturbances.

8.Right sided weakness more common than left sided weakness.

9.Hemiparesis more common than hemiplegia.

10.Motor aphasia most common type of aphasia in our study.

11.Smoking and Alcohol are the most common risk factor for ischemic stroke followed by Dyslipidemia, Diabetes mellitus and Hypertension.

12.Atrial fibrillation is the most common risk factor for embolic stroke.

13.Rheumatic heart disease is the most common cardiac disease causing embolic stroke.

14.Hypertension is the single most important factor for hemorrhagic stroke.

15.Vasculitis,Hypercoagulable states,Homocystinemia,APLA are rare risk factors for young stroke.

16.Weakness and speech disturbances were improved significantly.

17.Unsteadiness not improved significantly.

18.Death were seen in 12 cases

ABBREVIATIONS

ICH	-	Intra Cerebral Hemorrhage
SLE	-	Systemic Lupus Erythematosus
MCA	-	Middle Cerebral Artery
ACA	-	Anterior cerebral Artery
PCA	-	Posterior cerebral artery
PFO	-	Patent Foramen Ovale
CAD	-	Coronary artery disease
TIA	-	Transient ischemic attack
DYS	-	Dyslipidemia
HGIC	-	Hemorrhagic stroke
RHD	-	Rheumatic Heart disease
ASD	-	Atrial Septal Defect
CHD	-	Congenital Heart Disease
HT	-	Hypertension
CN	-	Cranial nerves
MBI	-	Modified Barthel Index
MRS	-	Modified Rankin Scale
CVT	-	Cerebral Venous Thrombosis
ASA	-	Atrial septal aneurysm
PVD	-	Peripheral vascular disease

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Study on Etiopathogenesis, Clinical presentation and outcome in 100 young stroke patients

Proforma

Name : Age : Sex : Wd:

Handedness : Marital status : Education:

Admitted with H/o

Past History : 1. Diabetes ☐ 2. Hypertension ☐

3. Hyperlipidemia ☐ 4. Migraine ☐

5. Cardiac disease ☐ 6. Collagen vascular disease ☐

7. Previous H/o TIA, Stroke ☐

Family History : DM / HT / CVA / CAD

Personal History : Smoking : ☐ Alcohol : ☐

Substance abuse : ☐ OCP intake : ☐

Sexual History : H/o Extra marital contact

Clinical Examination :

Symptoms

Visual defect :

Aphasia type :

Headache :

Seizures :

Weakness :

Unsteadiness:

General Examination :

Anemia : Carotid thrill or Bruit :

JVP : Pedal edema: Rashes :

Joint Deformities :

Vitals : PR ; BP : RR :

Fundus :

CNS : Higher functions : MMSE : / 30

Cranial nerve examination :

Spinomotor system :

Sensory system :

Cerebellum examination :

Spine and Cranium :

Other system :

CVS : RS : Abd :

Investigations :

(F) Bl.Sugar : (PP) Bl.Sugar : (R) Bl.Sugar :

(F) Lipid profile :

ECG : ECHO :

CT Brain :

MRI Brain / MRA / MRV :

Carodid vertebral Doppler :

X ray chest – PA view :

HIV 1 & 2 : Bl. VDRL :

Hypercoagulable status workup :

Protein C, S, Antithrombin III,

Igm Anticardiolipin AB :

Vasculitis work up :

Complete hemogram, ANA, ANCA, Rheumatoid factor

MASTER CHART

S.No.	NAME	Age	Sex	diagnosis	Smoking	Alcohol	HT	DM	IHD	Dyslipidemia	ECG	ECHO	BMI	ANA	RF	APLA	S.No.	Homocyst	VDRL	HIV	Xray chest	USG ABD	Carotid Doppler	CTBrain / MRI brain	Family H/o	Clinical presentation	ETIOLOGY/FINAL DIAGNOSIS
1	Karuppiah	40	M	CVA Rt hemiparesis	+	+	+	-	-	-	LVH	Con LVH	30	-	-	-	1	-	-	-	N		atheroma MCA	Lt MCA infarct	-	Aphasia& weakness	DM/HT/Smoking/alcohol Thrombotic stroke
2	Vani	32	F	CVA Lt hemiparesis	-	-	-	-	-	-	Tachycardia sinus	Cardiomyopathy	26	+	+	-	2	-	-	-	N	N	N	Rt MCA infarct	-	Weakness	ANA/RF/ Vasculitis.Thrombotic stro
3	Rajendran	44	M	CVA Rt hemiparesis	-	-	-	-	-	-	LAE AF	RHD MS/MR	22	-	-	-	3	-	-	-	straightening	N	N	Lt MCA infarct		Aphasia	RHD MS MR AF EMBOLIC STROKE
4	Karuppasamy	44	M	Postr. Circulation stroke	+	+	+	+	-	+	Tachycardia	LVH poor EF	32	-	-	-	4	-	-	-	Cardiomegaly	N	Atherosclerosis plague in vertebral artery	PICA occlusion		Un steadiness	DM/HT/Obesity/smoking / alcohol THROMBOTIC STROKE
5	Manickam	45	M	CVA Rt hemiparesis	-	-	-	-	-	-	N	N	22	-	-	-	5	+	-	-	N	N	N	Lt MCA infarct	+	aphasia weakness	Homocystinemia THROMBOTIC STROKE
6	Suseela	30	F	CVA Rt hemiparesis	-	-	-	-	-	-	N	N	22	-	-	+	6	-	-	-	N	N	N	Lt MCA infarct		aphasia weakness	APLA THROMBOTIC STROKE
7	Maruthiah	39	M	CVA Lt hemiparesis	+	+	-	-	-	-	N	N	36	-	-	-	7	-	-	-	N	N	N	ACA Rt infarct		Weakness	Obesity / smoking/ Alcohol - THROMBOTIC
8	Bharathi	30	F	CVA Rt hemiparesis	-	-	-	-	-	-	LAE Tachycardia	RHD MS	29	-	-	-	8	-	-	-	N	N	N	ACA Lt infarct		Weakness	RHD / MS /AF/EMBOLIC
9	Karthiga	36	F	CVA Lt hemiparesis		-	-	-	-	-	N	N	32	-	-	-	9	-	-	-	N	N	N	Rt MCA infarct		Weakness	Obesity THROMBOTIC STROKE
10	Ponraj	45	M	Postr. Circulation stroke	+	+	+	+	+	+	N	CAD	35	-	-	-	10	-	-	-	N	N	N	Bi cerebellar infarct		Un steadiness	HT/DM/IHD/Dyslipidemia THROMBOTIC
11	Adaikalam	37	M	CVA LT Hemiplegia	+	+	+	-	-	-	LVH	Con LVH	33	-	-	-	11	-	-	-	N	adrenal tumour	N	lCH in capsulo ganglionic bleed		Headache LOC	HT / smoking alcohol HGIC STROKE
12	Mookkammal	32	F	CVA R Hemiplegia	-	-	+	-	-	-	LVH	Con LVH	32	-	-	-	12	-	-	-	N	RAS Lt	N	Rt capsular gang. Bleed		LOC	HT/RAS HGIC STROKE
13	Pothumponnu	40	F	CVA Rt hemiparesis	-	-	+	+	-	+	N	N	32	-	-	-	13	-	-	-	N	N	ather.sclep laque in CCA	Lt MCA infarct		Weakness	DM / HT -THROMBOTIC
14	Raja Sri	44	F	Postr. Circulation stroke	-	-	+	+	-	+	N	N	34	-	-	-	14	-	-	-	N	N	ateroma SCA	Rt cerebellar infarct		Un steadiness	DM/HT THROMBOTIC STROKE

15	Rajathi	41	F	CVA Rt hemiparesis	-	-	-	-	-	-	RBBB	CHD ASD	24	-	-	-	15	-	-	-	Cardiomegaly	N	N	Lt MCA infarct		Weakness	CHD / ASD EMBOLIC STROKE
16	Arumugam	30	M	CVA Lt hemiparesis	-	-	-	-	-	-	LAE	RHD MS	26	-	-	-	16	-	-	-	LtHeartstraight	N	N	Rt MCA infarct		aphasia	RHD/MS/AF EMBOLIC STROKE
17	Thirupathi	37	M	CVA Rt hemiparesis	+	+	-	+	-	-	N	N	**35**	-	-	-	17	-	-	-	**N**	**N**	**N**	Lt MCA infarct	+	aphasia motor	DM / smoking / alcohol THROMBOTIC STROKE
18	Malairaj	43	M	CVA Rt hemiparesis	+	+	-	+	+	+	Old AW MI	CAD	32	-	-	-	18	-	-	-	Cardiomegaly	N	Plague at orgin of MCA	Lt MCA infarct	+	aphasia globa weakness	DM / smoking / alcohol THROMBOTIC STROKE
19	Senthilvel mani	43	F	CVA Lt Hemiplegia	-	-	-	-	-	-	AF RAE	CHD ASD	22	-	-	-	19	-	-	-	N	N	Normal	Rt MCA infarct		weakness	CHD/ASD/EMBOLIC STROKE
20	Vinodha	22	F	CVA Lt hemiparesis	-	-	-	-	-	-	Normal	Normal	22	+	+	-	20	-	-	-	N	N	Normal	Rt MCA infarct		weakness	CVA Rt Hemiparesis vasculitis
21	Ragavendran	29	M	CVA Lt hemiparesis	+	+	+	-	-	-	LVH	Con LVH	32	-	-	-	21	-	-	-	N	CRF	atheroma MCA	Rt MCA infarct		weakness	HT / smoking alcohol THROMBOTIC STROKE
22	Malaichamy	38	M	CVA Rt hemiparesis	+	+	-	-	-	-	N	N	34	-	-	-	22	-	-	-	N	N	Atheroma Lt MCA	Lt MCA infarct	+	aphasia weakness	Smoing / alcohol THROMBOTIC STROKE
23	Kannagi	43	F	CVA Lt hemiparesis	-	-	+	-	-	-	LVH	Con LVH	35	-	-	-	23	-	-	-	N	N	Atherosclero plaque in MCA	Rt MCA infarct		weakness	HT THROMBOTIC STROKE
24	Vetrivel	44	M	CVA Rt hemiplegia	+	+	+	-	-	-	N	N	29	-	-	-	24	-	-	-	N	N	N	Lt capsulo ganglion bleed		LOC	HEMOPHILIA HGIC STROKE
25	Ramu	45	M	CVA Lt Hemiplegia	-	-	-	-	-	-	RBBB AF	CHD ASD	24	-	-	-	25	-	-	-		N	N	Rt MCA infarct		aphasia sensory	CHD - ASO OS AF EMBOLIC STROKE
26	Murugan	38	M	Postr. Circulation stroke	+	+	-	+	-	+	Infr.wall ischemia	N	33	-	-	-	26	-	-	-		N	atheroma pica	Rt cerebellar infarct		unsteadiness	Smoing / alcohol/dys THROMBOTIC STROKE
27	Malathi	42	F	CVA Lt Hemiplegia	-	-	-	-	-	-	N		29	-	-	-	27	-	-	-		N	N	transverse sinus thrombu		weakness	CVT
28	Maruthi	40	F	CVA Rt hemiplegia	-	-	-	-	-	-	N	N	22	-	-	-	28	-	-	-	N	N	N	Transverse sinus thrombu		weakness	CVT
29	Malligai	17	F	CVA Rt hemiplegia	-	-	-	-	-	-	Bradycardia AF	RHD / MS / AF	22	-	-	-	29	-	-	-	N	N	N	Lt MCA infarct		aphasia weakness	RHD MS AF EMBOLIC STROKE
30	Maruthayee	38	F	CVA Lt Hemiplegia	-	-	-	-	-	-	Tachycardia	N	32	-	-	-	30	-	-	-	N	N	N	SSS thrombosis	+	seizures weakness	CVT
31	Manickam	45	M	Postr. Circulation stroke	+	+	+	+	+	+	Old infr wall MI	CAD	34	-	-	-	31	-	-	-	N	N	Atheroma vertebral artery	Lt PCA occlusion		unsteadiness	DM/HT/DYS THROMBOTIC STROKE
32	Vendhen	44	M	CVA Rt hemiplegia	+	+	-	+	-	-	N	N	29	-	-	-	32	-	-	-	N	N	Atheromo Lt MCA	Lt MCA infarct	+	aphasia global	DM/SMOKING/ALCOHOL THROMBOTIC STROKE
33	Kannan	42	M	Postr. Circulation stroke	**+**	**+**	**+**	-	-	-	LVH	Con LVH	30	-	-	-	33	-	-	-			**atherom a SCA**	Lt cerebellar infarct		unsteadiness	Smoking / alcohol / HT THROMBOTIC STROKE

34	Kannadasan	33	M	CVA Lt Hemiplegia	+	+	+	-	-	+	LVH	Con LVH	32	-	-	-	34	-	-	-	N	adrenal tumour	N	Rt capsulo ganglion bleed		weakness aphasia	HT HGIC STROKE
35	Shenbagam	32	F	CVA Rt hemiparesis	-	-	-	-	-	-	N	N	24	+	-	-	35	-	-	-	N	N	N	Lt MCA infarct		aphasia motor	VASCULITIS
36	Valli	31	F	CVA Rt hemiparesis	-	-	-	-	-	-	N	N	25	-	-	+	36	-	-	-	N	N	N	Lt transverse sinusthrombosi		Headache	CVT
37	Muthu	30	F	CVA Lt hemiparesis	-	-	-	-	-	-	N	N	23	-	-	-	37	-	-	-	N	N	N	SSS thrombosis		weakness	CVT
38	Veeran	42	M	Postr. Circulation stroke	+	+	+	+	-	+	N	N	33	-	-	-	38	-	-	-	N	N	PICA Atheroma	Lt PCA occlusion		Unsteadiness	DM/HT/Obesity/Dyslip THROMBOTIC STROKE
39	Samuvel	24	M	CVA Lt hemiparesis	-	-	-	-	-	-	RBBB	RHD ASD	24	-	-	-	39	-	-	-	N	N	N	Rt MCA infarct		Weakness	ASD EMBOLIC STROKE
40	Mayan	41	M	CVA Lt Hemiplegia	+		-	-	-	-	N	N	34	-	-	-	40	-	-	-	N	N	N	SSS thrombosis	+	weakness	Smoking/ALCOHOL CVT
41	Mohan	44	M	CVT Rt Hemiparesis	+	+	-	-	-	-			30	-	-	-	41	-	-	-	N	N	Atheroma MCA	Lt MCA infarct		weakness	Smoking alcohol THROMBOTIC STROKE
42	Ranjani	21	F	CVT Rt Hemiparesis	-	-	-	-	-	-	N	N	24	+	-	-	42	-	-	-	N	N	N	Lt MCA infarct		weakness	VASCULITIS
43	Maruthamuthu	41	M	CVA Lt Hemiplegia	+	+	-	-	-	+	N	N	33	-	-	-	43	-	-	-	N	N	N	Rt MCA infarct		LOC	Smoking / alcohol / THROMBOTIC STROKE
44	Kattupatti	25	M	CVA Lt Hemiplegia	-	-	-	-	-	-	AF	RHD MS MR	30	-	-	-	44	-	-	-	N	N	N	Rt MCA infarct		LOC	RHD MS MR EMBOLIC STROKE
45	Vasanthi	22	F	CVA Lt Hemiplegia	-	-	-	-	-	-	N	N	28	-	-	-	45	-	-	-	N	N	N	ssS thrombosis		weakness seizures	CVT
46	Vaijayanthi	24	F	CVA Rt hemiplegia	-	-	-	-	-	-	AF	RHD /MS/ AF	22	-	-	-	46	-	-	-	N	N	N	Lt MCA infarct		weakness	RHD / AF EMBOLIC STROKE
47	Banu	30	F	CVA Rt hemiparesis	-	-	-	-	-	-	N	N	22	-	-	-	47	-	-	-	N	N	N	SSS thrombosis		Headache	CVT
48	Senthil	43	M	CVA Rt hemiplegia	+	+	+	-	-	+	LVH	Coarction of aorta	32	-	-	-	48	-	-	-	N	N	N	Lt capsulo ganglion bleed		LOC	HT/ HGIC STROKE
49	Velavan	44	M	Postr. Circulation stroke	**+**	**+**	**+**	**+**	**+**	**+**	Inf.wall ischemia	**Cardiomyopathy**	32	-	-	-	49	-	-	-	N	N	Atheroma plaque vertebral	Lt cerebellar infarct		Unsteadiness	DM/ HT / IHD / Dyslip THROMBOTIC STROKE
50	Shenthamil	30	F	CVA Rt hemiplegia	-	-	-	-	-	-	AF	RHD MR/MVP	30	-	-	-	50	-	-	-	N	N	N	Lt MCA infarct		aphasia weakness	RHD MR EMBOLIC STROKE
51	Thenmozhi	31	F	CVA Rt hemiplegia	-	-	-	-	-	-	N	N	30	-	-	-	51	-	-	-	N	N	N	sss thrombosis		weakness seizures	CVT
52	Kanmani	43	F	CVA Rt hemiparesis	-	-	-	-	-	-	AF	RHD MS/MR	23	-	-	-	52	-	-	-	N	N	N	Lt MCA infarct		aphasia sensory	RHD / MS / MR EMBOLIC STROKE

53	Adaikalam	44	M	CVA Rt hemiplegia	+	+	-	+	+	+	Int.wall ischemia	Cardiomyopathy	32	-	-	-	53	-	-	-	N	N	N	Lt MCA infarct		aphasiaglobal weakness	DM/SM/AL/DYS/ EMBOLIC STROKE
54	Chendrayan	41	M	CVA Lt hemiparesis	+	+	-	-	-	-	N	N	30	-	-	-	54	-	-	-	N	N	N	Rt ACA infarct	+	weakness	SMOKING/ALCOHOL THROMBOTIC STROKE
55	Chelian	42	M	Postr. Circulation stroke	+	+	+	-	-	-	N	N	31	-	-	-	55	-	-	-	N	N	atheroma pica	Rt cerebellar infarct	+	unsteadiness	SMOKING/ALCOHOL/HT THROMBOTIC STROKE
56	Kandamuthu	41	M	CVA Rt hemiplegia	+	+		+		+	N	N	34	-	-	-	56	-	-	-	N	N	N	Lt MCA infarct		Sensory aphasia	SM/ALC/DM/DYS/ THROMBOTIC STROKE
57	Parvathy	30	F	CVA Rt hemiplegia	-	-	+	-	-	-	LVH	Con LVH	30	-	-	-	57	-	-	-	N	N	N	Lt capsulo ganglion bleed		headache/apha sia/weakne	HT HGIC STROKE
58	Iyyammal	42	F	CVA Lt Hemiplegia	-	-	+	-	-	-	LVH	Con LVH	32	-	-	-	58	-	-	-	N	CRF	N	Rt capsulo ganglion bleed		Weakness	HT / CRF HGIC STROKE
59	Mohamed Ali	44	M	Postr. Circulation stroke	+	+	+	+	+	+	Infr.wall ischemia	CAD	36	-	-	-	59	-	-	-	N	N	Atheroma plaque vertebral	Rt PICA occlusion		unsteadiness	DM/HT/SM/AL/IHD /DYS THROMBOTIC STROKE
60	Madhavan	30	M	CVA Rt hemiplegia	-	-	-	-	-	-	AF	CHD ASD	26	-	-	-	60	-	-	-	N	N	N	Lt MCA infarct		weakness aphasia motor	ASD/AF EMBOLIC STROKE
61	Kottaisamy	32	M	CVA Lt Hemiplegia	-	-	-	-	-	-	AF	RHD / MS	23	-	-	-	61	-	-	-	N	N	N	Rt MCA infarct		weakness	RHD / MS EMBOLIC STROKE
62	Balu	31	M	CVA Rt hemiplegia	-	-	-	-	-	-	AF	RHD / MS	23	-	-	-	62	-	-	-	N	N	N	Lt MCA infarct		aphasia weakness	RHD / MS / EMBOLIC STROKE
63	Lakshman	44	M	CVA Rt hemiplegia	+	+	-	+	-	-	N	N	34	-	-	-	63	-	-	-	N	N	N	Lt MCA infarct	+	aphasiaglobal weakness	DM/HT/SM/AL THROMBOTIC STROKE
64	Selvaraj	34	M	CVA Rt hemiplegia	+	-	+	-	-	-	LVH	Con LVH	30	-	-	-	64	-	-	-	N	CRF	N	Lt MCA infarct		aphaia global weakness	HT/SM/ CRF THROMBOTIC STROKE
65	Sattanathan	44	M	Postr. Circulation stroke	**+**	**+**	**+**	-	**+**	**+**	Old Ant wall MF	**CAD**	28	**-**	**-**	**-**	65	**-**	**-**	**-**	N	**N**	Atheroma in vertebral artery	Rt cerebellar infarct	+	unsteadiness	DM/HT/AL THROMBOTIC STROKE
66	Pappu	42	M	CVA Rt hemiplegia	-	-	+	-	-	-	LVH	Con LVH	29	**-**	**-**	**-**	66	**-**	**-**	**-**	N	CRF	N	Lt CApsulo ganglion bleed		weakness	HT / CRF HGIC STROKE
67	Ptichai	40	F	CVA Rt hemiplegia	+	+	+	+	-	-	N	N	33	**-**	**-**	**-**	67	**-**	**-**	**-**	N	**N**	atheroma mca	Lt MCA infarct		aphasiaglobal weakness	SM/AL/HT/DM THROMBOTIC STROKE
68	Stellamery	41	F	CVA RT hemiparesis	-	-	+	-	-	-	LVH	Con LVH	32	**-**	**-**	**-**	68	**-**	**-**	**-**	N	**N**	atheroma mca	LT MCA infarct		weakness	HT/ THROMBOTIC STROKE
69	Dharmaraj	44	M	CVA Lt Hemiplegia	+	+	+	-	-	-			34	**-**	**-**	**-**	69	**-**	**-**	**-**	N	**N**	atheroma carotid	R ACA infarct		weakness	SM/ALC/HT THROMBOTIC STROKE
70	Thangavel	43	M	Postr. Circulation stroke	+	+	+	-	-	-	N	N	34	**-**	**-**	**-**	70	**-**	**-**	**-**	N	**N**	Atheroma in PICA	Lt PICA occlusion		unsteadiness	SM/AL/HT THROMBOTIC STROKE
71	Arumugam	21	M	CVA Rt hemiparesis	-	-	-	-	-	-	AF Bradycardia	RHD MS/PHT	22	**-**	**-**	**-**	71	**-**	**-**	**-**	N	**N**	N	Lt MCA infarct		aphasiamotorw eakness	RHD/MS/AF EMBOLIC STROKE

72	Augustin	45	M	CVA Lt Hemiplegia	+	+	+	+		+	LVH	Con LVH	33	-	-	-	72	-	-	-	N	**N**	N	Rt ACA infarct		weakness	SM/AL/HTDM/IHD THROMBOTIC STROKE
73	Marimuthu	21	M	CVA Rt hemiparesis	-	-	-	-	-	-	N	CHD ASD	28	-	-	-	73	-	-	-	N	**N**	N	Lt MCA infarct		weakness	ASD EMBOLIC STROKE
74	Manimari	29	F	CVA RT Hemiplegia	-	-	+	-	-	-	LVH	Con LVH	30	-	-	-	74	-	-	-	N	**CRF**	N	LT caps gang bleed		weakness	HT / CRF HGIC STROKE
75	Kaleeswari	25	F	CVA Rt hemiparesis	-	-		-	-	-	AF Bradycardia	RHD MS/PHT	23	-	-	-	75	-	-	-	N	**N**	N	Lt MCA infarct		motor aphasia	RHD/MS/AF EMBOLIC STROKE
76	Sundaram	25	M	CVA RT Hemiplegia	-	-	+	-	-	+	LVH	Con LVH	28	-	-	-	76	-	-	-	N	**N**	N	L putaminal hge		LOC weakness	HT / CRF HGIC STROKE
77	Vinodhkumar	41	M	CVA Rt hemiplegia	+	+	+	+	-	-	N	N	34	-	-	-	77	-	-	-	N	**N**	atheroma carotid	Lt ACA infarct	+	Weakness	DM/HT/SM/AL THROMBOTIC STROKE
78	Suresh	44	M	CVA Rt hemiplegia	+	+	+	-	-	-	N	N	30	-	-	-	78	-	-	-	N	**N**	atheroma carotid	Lt ACA infarct	+	weakness	SM/AL/HT THROMBOTIC STROKE
79	Aravindh	32	M	CVA Rt hemiparesis	-	-	-	-	-	-	AF	RHD MS	26	-	-	-	79	-	-	-	N	**N**	N	Lt MCA infarct		Sensory aphasia	RHD/MS/AF EMBOLIC STROKE
80	Kannaiah	43	M	CVA Lt hemiparesis	+	+	+	-	-	-	LVH	Con LVH	24	-	-	-	80	-	-	-	N	**N**	N	Rt capsulo ganglion bleed		Weakness	HT/ HGIC STROKE
81	Mokkasamy	42	M	Postr. Circulation stroke	+	+	+	+	-	-	N	N	32	-	-	-	81	-	N	N	N	N	atheroma pica	Lt cerebellar infarct	-	Unsteadiness	SM/AL/HT/DM THROMBOTIC STROKE
82	Sundarmenan	34	M	CVA Rt hemiplegia	-	-	+	-	-	-	LVH	Con LVH	30	-	-	-	82	-	N	N	N	CRF	N	Lt MCA infarct	-	weakness	HT/CRF HGIC STROKE
83	Banumathy	28	F	CVA Rt hemiplegia	-	-	-	-	-	-	LAE AF	RHD / MS	26	-	-	-	83	-	N	N	N	N	N	Lt MCA infarct	-	weakness	RHD/MS EMBOLIC STROKE
84	Mannan	33	M	CVA Rt hemiplegia	-	-	-	-	-	-	AF	RHD / MS	27	-	-	-	84	-	N	N	N	N	N	Lt MCA infarct	-	weakness	RHD/MS EMBOLIC STROKE
85	Chellam	21	F	CVA Rt hemiplegia	-	-	-	-	-	-	N	N	32	+	-	-	85	-	N	N	N	N	N	SSS thrombosis		weakness Seizures	CVT
86	Kannammal	28	F	CVA Rt hemiplegia	-	-	-	-	-	-	N	N	30	-	-	-	86	-	N	N	N	N	N	SSS thrombosis	-	convulsions weakness	CVT
87	Mookiah	45	M	CVA Lt Hemiplegia	+	+	+	-	-	+	LVH	Con LVH	26	-	-	-	87	-	N	N	N	N	N	ICH Rt putamen	-	LOC	HT HGIC STROKE
88	Sangumani	42	M	CVA RT Hemiplegia	-	-	-	-	-	-	AF	RHD / MS	22	-	-	-	88	-	N	N	N	N	N	Lt MCA infarct	-	seizures	RHD/MS EMBOLIC STROKE
89	Balaji	37	M	CVA Rt hemiplegia	+	+	+	-	-	+	LVH	Con LVH	29	-	-	-	89	-	N	N	N	N	atheroma mca	Lt MCA infarct	-	aphasia weakness	HT / AL THROMBOTIC STROKE
90	Manimaran	41	M	CVA Lt Hemiplegia	-	-	-	-	-	-	LVH	RHD / MR	27	-	-	-	90	-	N	N	N	N	N	Lt MCA infarct	-	aphasia weakness	RHD/MR EMBOLIC STROKE

91	Ponnumani	43	M	CVA Rt hemiplegia	-	-	+	-	-	-	LVH	Con LVH	33	-	-	-	91	-	N	N	N	N	atheroma mca	Lt MCA infarct	-	hemianasthesia	HT/ THROMBOTIC STROKE
92	Avudaiammal	43	F	CVA Rt hemiplegia	-	-	+	+	+	+	CAD	CAD	26	-	-	-	92	-	N	N	N	N	N	Lt MCA infarct	-	weakness	CARDIOMYOPATHY EMBOLIC STROKE
93	Sumanth	45	M	CVA Lt hemiparesis	-	-	+	-	-	-	LVH	Con LVH	27	-	-	-	93	-	N	N	N	N	N	Rt capsulo gang bleed	-	headache weakness	HT HGIC STROKE
94	Sundaram	17	M	CVA Rt Hemiplegia	-	-	-	-	-	-	AF	RHD / MS/MR	26	-	-	-	94	-	N	N	N	N	N	Lt mCA infarct	-	weakness	RHD/MS/MR EMBOLIC STROKE
95	Pandian	44	M	CVA Rt hemiplegia	+	+	+	+		+	LVH	Con LVH	33	-	-	-	95	-	N	N	N	N	atheroma mca	Lt MCA infarct	-	weakness	SM/AL/HT/DM/ THROMBOTIC STROKE
96	Ponnuthai	32	F	CVA Rt hemiplegia	-	-	-	-	-	-	Tachycardia	N	26	+	+	-	96	-	N	N	N	N	N	transverse sinus thrombu	-	weakness seizures	CVT
97	Mayandi	34	M	CVA Lt hemiplegia	+	+	+	-	-	-	LVH	Con LVH	30	-	-	-	97	-	N	N	N	RAS	N	Rt capsulo ganglion bleed	-	LOC	HT HGIC STROKE
98	Virumandi	44	M	CVA Rt hemiplegia	+	+	+	+	-	-	N	N	30	-	-	-	98	-	N	N	N	N	atheroma carotid	Lt ACA infarct	-	Weakness	HT/DM/AL/SM THROMBOTIC STROKE
99	Sathyan	43	M	CVA Lt hemiparesis	+	+	-	-	-	-	N	N	36	-	-	-	99	-	N	N	N	N	atheroma R ICA	Rt MCA infarct		Weakness	SM/AL THROMBOTIC STROKE
##	Mariammal	30	F	CVA Rt hemiparesis	-	-	-	-	-	-	AF	RHD MS	36	-	-	-	100	-	N	N	N	N	N	Lt MCA infarct		Weakness aphasia	RHD/MS EMBOLIC STROKE

Ref. No. 20735/E4/2/2013 Govt. Rajaji Hospital,
Madurai.20. Dated: .12.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for November 2013
Approved list -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 18.11.2013, Monday at 10.00 am to 12.00.noon at the Anaethesia Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. I. Jeyaraj, M.S., (Anatomy) Cell.No 9566211947	Director & Professor Institute of Anatomy /V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048	Professor of Medicine Madurai Medical College	Member
6. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031	Professor & H.O.D of Surgery i/c Madurai Medical College	Member
7. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
8. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
9. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Project was approved by the committee

..2..

Name of P.G.	Course	Name of the Project	Remarks
Dr. S. Senthur Raja Pandian	PG in DM (Neurology), Madurai Medical College and Government Rajaji Hospital, Madurai.	Etiopathogenesis, clinical presentation, and outcome in 100 young stroke patients.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

20/12/13

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-thro. Head of the Department concerned

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STUDY ON ETIOPATHOGENESIS, CLINICA PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS

Dissertation submitted
In partial fulfillment of the regulation for
the final examination of

BRANCH - I
D.M.(NEUROLOGY)
AUGUST 2014